BRAIN.

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Original Articles and Clinical Cases.

ON THE LOCALISATION OF INTRACRANIAL TUMOURS.[1]

BY BYROM BRAMWELL, M.D.

At a discussion on Intra-cranial Surgery at the Edinburgh Medico-Chirurgical Society a few years ago (February 21, 1894), I analysed all the cases of intracranial tumour—82 in number—which had been under my own care up to that time. Since being invited to take part in the discussion, I have carefully gone over the notes of these cases, weeding out a few cases in which the diagnosis was perhaps doubtful, and adding the additional cases which have since come under my notice.

The *total number* of cases of intracranial tumour which have been under my own care during life up to the present time is 122.

The *results* in these 122 cases were as follows:—

Fatal:—

(*a*) With *post-mortem* examination	40		
(*b*) Without *post-mortem* examination	36		
	—	...	76

Cured or apparently cured:—

(*a*) Non-syphilitic cases (some of them were perhaps cases of distended ventricles and not tumours)	4		
(*b*) Syphilitic cases	7		
	—	...	11

[1] Being a contribution to the discussion on Tumours, published in BRAIN, 1898, p. 291.

Greatly relieved :[1]—			
(*a*) Non-syphilitic cases	1		
(*b*) Syphilitic cases	10		
	—	...	11
Remaining in statu quo :—			
(*a*) Non-syphilitic cases	12		
(*b*) Syphilitic cases	1	...	13
Result unknown :—	11		
	—	...	11
Total		...	122

In this discussion I shall only, of course, refer to the cases in which the exact localisation of the tumour was definitely ascertained by *post-mortem* examination, and to two cases, in one of which the position of the tumour was verified, and in the other not verified by operative procedure.

In the following table, the position and nature of the tumour in the 40 cases which were verified by *post-mortem* examination are shown :—

Table showing Position and Nature of the Tumour in 40 Cases of Intracranial Tumour under Dr. Byrom Bramwell's care during life, and examined Post mortem.

No.	Age.	Sex.	Position of Tumour.	Nature of Tumour.
1	41	M.	Right angular gyrus and adjacent parts of the parietal and occipital lobes.	Syphilitic.
2	40	M.	Right frontal lobe, first convolution and subjacent white matter.	Glioma and cyst.
3	45	M.	Left frontal lobe, first convolution and subjacent white matter.	Lympho - sarcomatous cyst.
4	64	F.	Left lateral lobe of the cerebellum.	Sarcoma of the membranes.
5	34	M.	Right frontal lobe, first and second convolutions and subjacent white matter.	Syphilitic.
6	20	F.	Right motor area and second and third frontal convolutions, island of Reil, and tip of temporo-sphenoidal lobe.	Sarcoma of the membranes.
7	5	M.	Six tumours: (1) first left frontal convolution; (2) tip of left occipital lobe; (3) right gyrus fornicatus: (4) left gyrus fornicatus; (5) right angular gyrus; (6) head of left corpus striatum.	Tubercular.
8	9	F.	Cerebellum, right lateral lobe.	Tubercular.
9	34	M.	Right motor area.	Secondary cancer.

[1] In several of these cases the ultimate result is not known.

No.	Age.	Sex.	Position of Tumour.	Nature of Tumour.
10	1½	F.	Left crus and cerebellum.	Tubercular.
11	60	M.	Left occipital lobe.	Syphilitic.
12	4	M.	Vertex.	Perforating sarcoma.
13	50	M.	Right optic thalamus.	Glio-sarcoma.
14	39	M.	Motor area and subcortical white matter.	Glio-sarcoma.
15	18	M.	Cerebellum: four tumours—three in the left and one in the right lateral lobe.	Tubercular.
16	34	F.	Right side of pons.	Glio-sarcoma.
17	34	F.	Second left frontal convolution.	Glio-sarcoma.
18	63	F.	Left centrum ovale, just behind motor area and right island of Reil, and right lenticular nucleus.	Glio-sarcoma.
19	29	F.	Left motor area and posterior end of the third left frontal convolution, and the subjacent white matter.	Glioma with cyst.
20	54	M.	Right arm centre.	Glioma.
21	28	F.	Pons.	Glioma.
22	32	M.	Left frontal, temporo-sphenoidal and occipital lobes.	Glioma and cyst.
23	45	M.	Right centrum ovale, internal capsule and optic thalamus.	Glioma.
24	24	M.	Cerebellum, middle and right lateral lobes.	Glioma and cyst.
25	47	F.	Vertex.	Cancer of membranes.
26	47	F.	Left frontal lobe.	Glioma and cyst.
27	24	M.	Cerebellum, left lateral and middle lobes.	Glioma.
28	30	M.	Left centrum ovale and motor area.	Glioma.
29	17	F.	Cerebellum, middle and right lateral lobes.	Glioma.
30	51	F.	Base, pressing on left side of pons.	Fibro-sarcoma of membranes.
31	61	F.	Cerebellum, right lateral lobe, left lenticular nucleus and right ascending parietal convolution.	Secondary cancer.
32	26	F.	Left occipital cortex, etc.	Melanotic sarcoma.
33	21	F.	Pituitary body.	Sarcoma.
34	26	M.	Left centrum ovale.	Large glioma.
35	52	M.	Left frontal lobe.	Glioma.
36	40	M.	Base, right side of pons.	Fibro-sarcoma.
37	57	M.	Right temporo-sphenoidal lobe and second right frontal convolution.	Glioma and cyst.
38	8	F.	Left crus.	Tubercular.
39	42	M.	Left parietal lobe. Also large abscess in subjacent white matter.	Fibro-cellular.
40	7½	F.	Right side of pons, right crus and cerebellum.	Glioma.

On analysing this table I find that the position of the tumour in the 40 fatal cases which were examined *post mortem* was as follows:—

Frontal Lobe.—In six cases the tumour was confined to the frontal lobe (viz., 2, 3, 5, 17, 26 and 35).

In five other cases (viz., 6, 7, 19, 22 and 37) the frontal lobe was involved in addition to other parts.

Island of Reil.—In three cases (18, 19 and 22) the tumour involved the island of Reil; in all of these cases other parts were also involved.

Motor Area and Subcortical Motor Tracts.—In ten cases the tumour was situated in the motor area or subcortical tissues immediately below the motor area (viz., 6, 9, 14, 18, 19, 20, 23, 25, 28 and 34). In several of these cases other parts (frontal lobe, &c.) were also involved.

Tempero-Sphenoidal Lobe.—In two cases (viz., 22 and 37) the tumour was situated in the temporo-sphenoidal lobe; in both of these cases other parts were also involved.

Occipital Lobe.—In one case (11) the lesion (a healed syphilitic gumma) was limited to the occipital lobe; and in three cases (7, 22 and 28) the occipital lobe was involved as well as other parts.

Parietal Lobe.—In two cases (viz., 1 and 39) the tumour was situated in the parietal lobe.

Basal Ganglia. (*a*) *Optic Thalamus.*—In one case (13) the tumour had its primary seat in the right optic thalamus. In three other cases (22, 23 and 32) the optic thalamus was involved along with other parts.

(*b*) *Lenticular Nucleus.*—In two cases (18 and 31) the lenticular nucleus was involved along with other parts.

(*c*) *Caudate Nucleus.*—In two cases (7 and 18) the caudate nucleus was involved along with other parts.

Base of the Brain.—In three cases (30, 33 and 36) the tumour was situated at the base of the brain outside the nervous tissues.

In one of these cases (33) the tumour affected the *pituitary body.*

Crus Cerebri.—In two cases (10 and 33) the tumour was situated in the crus cerebri.

Pons Varolii.—In three cases (16, 21 and 40) the tumour was situated in the pons. In two other cases (30 and 36) the pons was implicated by tumours which had their primary seat outside the nervous tissues.

Cerebellum.—In seven cases (4, 8, 15, 24, 27, 29 and 31) the tumour was situated in the cerebellum. In three other cases (10, 32 and 40) the cerebellum was also involved by a

tumour, either springing from the membranes, or a tumour which involved other parts of the nervous tissues as well as the cerebellum.

Various Situations.—In two cases (7 and 32) the tumour or tumours involved a number of different parts of the cerebral and cerebellar tissues.

In one case (12) the tumour (a perforating sarcoma of the skull) involved the outer surface of the dura mater in the frontal and vertical regions and the frontal and parietal bones.

The primary seat of the tumour in the 40 cases was therefore as follows :—

	Cases
Frontal lobe	6
Motor area	10
Temporo-sphenoidal lobe	2
Occipital lobe	1
Parietal lobe	2
Optic thalamus	1
Base (extra nervous)	3
Crus cerebri	2
Pons Varolii	3
Cerebellum	7
Various positions	2
Extradural (perforating sarcoma of the skull) ...	1
Total ...	40

The number of cases included in any one group is too small to warrant generalised conclusions, especially since some of the cases were seen twenty-four years ago; but many of the individual cases afford important information as to the positive (localising) symptoms and as to the negative symptoms which may result from the presence of a tumour in the special parts of the brain and cerebellum which are included in this discussion. (I shall only, of course, refer to tumours in the frontal, temporo-sphenoidal and occipital lobes, the basal ganglia and the cerebellum, since tumours involving the motor area and the subjacent white matter, and tumours of the base, pituitary body, crus cerebri, pons Varolii and medulla oblongata are not included in this discussion.)

I propose, then, to detail briefly the more important clinical features of some of the individual cases, pointing out the positive and negative facts of localising value which they exhibit. The negative facts are in many instances very striking; and, from a diagnostic point of view, are, of course, as important as the positive localising symptoms. Speaking generally, I may say that as my experience of intracranial tumours increases I become more and more cautious in drawing conclusions from the clinical data as to the exact position of the new growth. I have met with cases in which an intracranial tumour has been situated in every one of the parts of the cerebrum which are specially included in this discussion, and in the cerebellum, in which there were absolutely no localising symptoms indicative of the position of the tumour. I make one exception only, for when the occipital lobe is the seat of the tumour (*i.e.*, when the half-vision centre or the optic radiations of Gratiolet are implicated by the tumour), hemianopsia is probably always present. But I speak tentatively on this point, for I have seen very few tumours of the occipital lobe.

In two cases (4 and 39) a tumour was found after death, the presence of which was entirely unsuspected during life, *i.e.*, in both cases the tumour seemed to be entirely "latent."

Tumours of the Frontal Lobe.

In six cases the tumour was limited to the frontal lobe, and in five other cases the frontal lobe was involved along with other parts. The more important features of the six cases in which the tumour was confined to the frontal lobe were as follows:—

(*A.*) Cases in which the Tumour was limited to the Frontal Lobe.

Case I. (Case 2 in the Table). *Glioma and cyst of the right frontal lobe involving the middle part of the first frontal convolution and the subjacent white matter.*

D. M., aged 40, pitman, admitted to the Newcastle-on-Tyne Infirmary on March 22, 1875.

A. General Symptoms. Headache (intense and usually frontal); vomiting (frequent and severe); giddiness; optic neuritis (intense, limited to the left eye, the right eye having been previously destroyed by an injury); several severe general epileptic fits.

B. Localising Symptoms. (*a*) *Mental Symptoms.* There is a marked change in the patient's mental condition; he has a silly expression; is at times irritable, obstinate and passionate, at other times depressed and stupid; his memory is markedly impaired; he frequently bursts into meaningless laughter; he sleeps well, but while sleeping grinds his teeth. Soon after his admission to hospital he developed delusions of suspicion and had to be sent to an asylum.

FIG. 1.

Section through the right frontal lobe of the brain in the case of D. M. (Case 1), showing the position of the lesion.

(*b*) *Motor Functions.* The gait is peculiar, he takes a few quick steps to the left, then a few quick steps to the right, often breaking into a short run; he frequently has attacks of temporary loss of power in the left side, occasionally in the right side; he every now and again falls, apparently from sudden loss of power in the left leg.

The *knee-jerks* were not examined; the *plantar reflex* was more marked on the left than on the right side.

(*c*) *Speech.* No aphasic symptoms.

(*d*) *Pseudo-localising Symptoms.* A cicatrix, the result of a severe head injury received a year ago, is present 2½ inches above and slightly behind the left ear; he was quite well until this injury; the head symptoms developed immediately

after the injury. Perhaps the tumour developed at the point of *contre-coup*.

Result. He became comatose on September 18, 1875, and died on September 20.

Post-mortem. Intra-cranial pressure greatly increased; Pacchionian bodies very prominent and had eroded the skull; convolutions flattened and sulci effaced; ventricles somewhat dilated; a gliomatous tumour and cyst in the right frontal lobe, involving the upper frontal convolution and the subjacent white matter (see fig. 1). Brain otherwise quite normal; weight, 59 ounces (cerebrum, 50½; cerebellum, 8½ ounces).

Remarks. In this case mental symptoms were very marked; and temporary paralysis, usually on the opposite side, sometimes in *both* legs, frequently recurred; the gait was peculiar; grinding of the teeth also occurred.

Case 2 (Case 3 in Table). *Glioma and cyst of the left frontal lobe, involving the middle part of the first frontal convolution and the subjacent white matter.*

W. A., aged 45, pitman, admitted to the Newcastle-on-Tyne Infirmary on October 1, 1894, suffering from an intra-thoracic tumour and head symptoms. The case was a very interesting and complicated one; the head symptoms were as follows:—

General Symptoms. No headache, vomiting, or giddiness mentioned in the notes; optic discs not examined; no fits.

Mental Symptoms. A marked change in patient's mental condition; is irritable and obstinate, will sit silent for hours and only answers questions in monosyllables; sleeps badly and often grinds his teeth.

Speech. Distinct motor aphasia.

Motor Functions. Right hemiplegia, which varied *greatly* in its degree from time to time.

Knee-jerks not examined.

Result. The mental symptoms and the paralysis varied considerably from day to day. A few days before death the right-sided hemiplegia, which the day previously had almost passed off, *suddenly* became complete, diarrhœa developed, the temperature rose to 102°, and the patient died November 6, 1894.

Post-mortem Appearances. Large lympho-sarcoma in thorax; pericarditis; secondary lympho-sarcomatous deposits in the abdominal and mesenteric glands, the left suprarenal capsule

and the small intestine; lympho-sarcomatous cyst the size of a hen's egg in the left frontal lobe, involving the middle part of the first frontal convolution and the subjacent white matter (see fig. 2); a quite recent embolus in the left middle cerebral artery (apparently this was the cause of the complete hemiplegia which developed suddenly a few days before death).

FIG. 2

Section through the left frontal lobe of the brain in the case of W. A. (Case 2), showing the position of the lesion.

Remarks. In this case mental symptoms were very marked; right hemiplegia very variable in degree and some motor aphasia seemed to be caused by the tumour; grinding of the teeth also occurred.

Case 3. (Case 5 in Table). *Syphilitic tumour involving the posterior end of the 1st and 2nd right frontal convolutions and the subjacent white matter and irritating the adjacent part of the motor cortex.*

J. M., aged 34, bricklayer, admitted to the Newcastle-on-Tyne Infirmary on November 4, 1875, suffering from left hemiplegia and Jacksonian epilepsy.

Previous History. Syphilis seven years previously. For two years "rheumatic" pains, worse at night; several attacks of severe headache. Seven weeks ago, a severe, general epileptic fit; two weeks ago, a second fit, unattended with loss of consciousness, limited to the left side and followed by left hemiplegia.

General Symptoms. Headache (severe, frontal and vertical); double optic neuritis, intense in degree.

Mental Symptoms. Mental condition very much impaired, all the mental processes being markedly blurred.

Speech. No aphasia.

Motor Functions. Left hemiplegia, involving the face, arm and leg, following Jacksonian epilepsy, which involved the left side of the body, and which had occurred a fortnight previously.

Knee-jerks not examined; *plantar reflex* more marked on the right (non-paralysed) side.

Immediate Result. Under anti-syphilitic treatment the patient made an excellent recovery and was discharged quite well on January 31, 1876.

Subsequent Progress. On September 23, 1876, he fell from a high scaffold, fractured his clavicle and received a severe cut on the right side of his head; was confined to bed for a month, suffering from severe headache; then returned to work.

On February 9, 1877, was seized with Jacksonian epilepsy; the fits continued to recur with great regularity and frequency; was re-admitted to the Newcastle-on-Tyne Infirmary on February 23, 1877, suffering from left hemiplegia and frequently recurring attacks of Jacksonian epilepsy; the spasms commenced with drawing up of *both* legs and chiefly affected the left side of the body; it was calculated that the patient had 4,320 fits.

Under treatment rapid improvement occurred, and the patient was discharged, well, on April 20, 1877.

He continued to follow his employment, and so far as could be ascertained made no complaint until December 31, 1877, when he was seized with a severe general epileptic fit, followed by coma and death.

Post-mortem Appearances. The scalp and skull-cap were natural; the dura was firmly adherent over a circumscribed spot corresponding to the posterior end of the second right frontal convolution. The surface of the brain at the point of adhesion was depressed, and on cutting into it this was found to be due to the contraction of a new growth (syphilitic gumma). The tumour involved the posterior end of the first and second right frontal convolutions and the subjacent white matter (see fig. 3); it did not directly involve the adjacent motor cortex (ascending frontal convolution).

Remarks. In this case the frequently recurring attacks of Jacksonian epilepsy, which commenced with drawing up

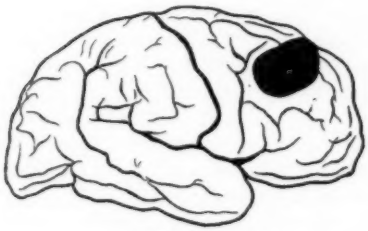

FIG. 3.

Right hemisphere of the brain in the case of J. M. (Case 3), showing the position of the lesion.

of *both* legs, suggested that the tumour was situated in the motor area (leg centre, or involving both leg centres at the vertex); the tumour involved the second right frontal convolution and its subjacent white matter; it had no doubt irritated the adjacent part of the motor area, but that was the arm centre.

Case 4. (Case 17 in Table). *Two sarcomatous tumours of the left frontal lobe; one small (the size of a Spanish olive) in the tip; the other large (the size of a large egg) was limited to, and had produced complete destruction of, the posterior end of the second left frontal convolution; no localising symptoms; no agraphia.*

J. S., aged 34, widow, admitted to the Edinburgh Royal Infirmary on February 15, 1896, suffering from headache, vomiting and dimness of vision.

Duration. The headache commenced a year ago; five months ago the patient experienced a tingling sensation all over the body (resembling that of the foot being asleep) and then vomited; has continued to vomit occasionally since; five weeks ago she noticed that her sight was becoming dim.

General Symptoms. Headache (very severe, occipital and frontal, continuous, with exacerbations, for the past five months); vomiting (occurs usually with exacerbations of the headache, generally before breakfast); dimness of vision in the left eye; giddiness and an occasional tendency to fall backwards when walking; a prickling sensation in the eyes; double vision;

double optic neuritis, more marked in the left than in the right eye, but moderate in degree even in the left eye.

Mental Symptoms. Absolutely none; the patient is intelligent and well educated, extremely bright and active mentally.

Speech. No aphasic defect of any kind; in particular, no agraphia.

Motor Functions. Slight paralysis of the left external rectus muscle and associated nystagmus in the right eye when the patient attempts to look right out to the left.

Knee-jerks equal and normal in degree.

Diagnosis. An intracranial tumour; position uncertain, but probably in the left hemisphere, because of the paralysis of the left external rectus.

Progress of the Case. During her stay in hospital the patient continued to have very severe headache and frequent attacks of vomiting.

On March 12, 1896, she complained of feeling dull and heavy; the optic neuritis was more marked.

On March 18, 1896, was discharged at her own request.

On June 1, 1896, was re-admitted; eyesight worse, optic neuritis more marked; headache intense, chiefly frontal; vomiting frequent and severe; giddiness frequent. Attacks of pain and a prickling feeling in the left side of the lips, tongue, roof of the mouth and left side of the face, never affecting the right side of these parts; occurring in paroxysms of some hours' duration; a numb feeling on the left side of the face, but no discoverable anæsthesia; the slightest touch is felt on the left side of the face, but the patient says the sensation produced by a slight touch is "awful different" from that produced by a corresponding touch on the right side. Occasional jerkings of both hands and arms, with a feeling of weakness in the legs and a sensation of nausea; these attacks occur in paroxysms of about five minutes' duration. Optic neuritis more marked. No mental or aphasic symptoms. The paralysis of the left external rectus *in statu quo.*

Knee-jerks equal and normal in degree.

June 9, 1896. Had an attack of jerkings affecting both arms and both legs and lasting for five minutes. The spasm commenced in the right leg.

June 10, 1896. Had an extremely severe attack of frontal headache; it developed suddenly, was attended with great restlessness and excitement, and was followed by extreme exhaustion; was only relieved by a hypodermic injection of morphia.

Several attacks of a similar nature, all requiring morphia for their relief, occurred during the next week.

June 17, 1896. Had an attack of clonic spasms (Jacksonian epilepsy) affecting the *left* foot and leg muscles.

June 22 *and* 26, 1896. Attacks of clonic spasms affecting both arms and both legs and the *left* side of the face.

June 30 *and July* 4. Attacks of intense headache, followed by great exhaustion; relieved by morphia hypodermically. These attacks were so alarming that palliative trephining (for the relief of the headache) was decided upon.

Up to this time, there had been absolutely no mental symptoms, no aphasic symptoms, in particular no agraphia.

The *operation* was performed by Mr. Cotterill on July 7, 1896, the trephine opening being made over the right motor area, since on June 17 the clonic spasms had been limited to the *left* foot and leg, though, in the earlier stages of the case, I was on the whole inclined to think that the tumour was probably situated on the right side. On opening the dura the intra-cranial pressure was found to be increased; nothing else abnormal was seen.

The operation was followed by great relief as regards the headache, but left hemiplegia and a condition of partial dementia unfortunately developed, evidently due to cerebritis.

The patient was re-admitted to my ward some three months later completely hemiplegic on the left side, and in a condition of marked mental enfeeblement. During the next two months there was some slight improvement.

On December 21, 1896, she was sent to the Rosslynlee Asylum. I am indebted to the Superintendent, Dr. Mitchell, and to his Assistant, Dr. Cross, for the following notes of her condition:—

On admission she was suffering from left hemiplegia and mental enfeeblement, and she was facile and emotional.

On February 1, 1897, she was better, talking sensibly, and very grateful for what was done for her.

On March 4, 1897, sensible and cheerful. Dr. Mitchell at this date wrote me:—"I cannot detect much wrong with her mentally, except mild enfeeblement and emotionalism."

During April, 1897, she became worse; her articulation and voice were much weaker; it was doubtful if she correctly understood what was said to her; her condition was that of dementia. She died on *May* 22, 1897.

On post-mortem examination the membranes were adherent

over the right motor area (the position of the trephine wound); the subjacent cortical and sub-cortical tissues were sclerosed—in a cicatricial condition, the result of meningo-cerebritis.

Two sarcomatous tumours were present in the left frontal lobe, viz.:—One of small size (the size of a Spanish olive) in the very tip. Another of large size (fully the size of a hen's egg) had replaced and entirely destroyed the posterior third of the second left frontal convolution (see fig. 4); the posterior end of the first left frontal convolution and the adjacent part of the left ascending frontal convolution were encroached upon but not destroyed; the third left frontal convolution did not appear to be involved.

The brain was otherwise normal. Dr. Cross, the Assistant Medical Officer to the Asylum, kindly brought the brain to me after the *post-mortem* examination, and I personally verified the exact localisation, as described and depicted by him.

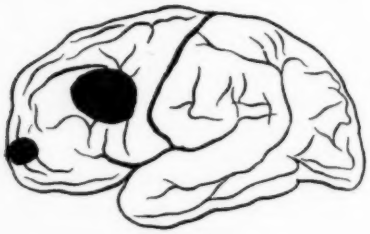

FIG. 4.

Left hemisphere of the brain in the case of J. S. (Case 4), showing the position of the tumours.

Remarks.—In this case, in which the posterior third of the second left frontal convolution (Exner's writing-speech centre) was entirely destroyed, there were, up to the time of the operation, absolutely no mental symptoms and no agraphia. While the patient was in hospital, she was in the habit of writing to her friends, and they state that there was absolutely no defect in her writing or in her power of expressing herself in writing. After the operation she did not write any letters (owing to the enfeebled mental condition), but she was able to write. While she was in the Rosslynlee Asylum, she gave instructions for

her will, and wrote with her own hand, "Please give my father-in-law M—— B——, my money," and signed this document.

In this case there were no symptoms indicative of the position of a tumour in the left frontal lobe. The following pseudo-localising symptoms (suggestive of a lesion in other parts of the brain) were observed during the course of the case:—A tendency to fall backwards; slight paralysis of the left external rectus muscle (probably due to indirect pressure on the left 6th nerve); a prickling feeling in the *left* side of the lips, tongue, roof of the mouth and face; occasional jerkings of *both* legs; localised clonic spasms affecting the muscles of the *left* foot and leg; spasmodic twitchings of the *left* side of the face.

Case 5. (Case 26 in Table). *Large glio-sarcomatous tumour and cyst of the left frontal lobe, involving and destroying the anterior two-thirds of the first and second and the anterior half of the third left frontal convolution and the subjacent white matter; the posterior third of the first and second and the greater part of the posterior end of the third left frontal convolutions and their subjacent white matter were softened, œdematous and to some extent infiltrated, though not completely destroyed by the new growth.*

A. B., female, single, aged 47, was seen with Dr. Foulis on October 16, 1884, complaining of headache, vomiting and partial loss of power in the right arm and leg.

Previous History.—At the age of 12 years, suffered from "brain fever"; at the age of 37, had an epileptic fit, after which a distinct and permanent change in her mental condition was noticed; at the age of 45, ceased to menstruate, and immediately after this the cerebral symptoms for which I saw her began to develop.

General Symptoms.—Headache (very severe, at first in the area of distribution of the left great occipital nerve, afterwards in the left frontal and parietal regions); vomiting; pseudo-apoplectic attacks (in which the patient would sleep for twenty-four hours at a time), attended with pyrexia (in one of them the temperature rose to 105°); no optic neuritis (though Dr. Thomas Barlow, who had seen the patient some months previously, had found slight optic neuritis in the left eye).

Mental Symptoms.—Vacant expression; loss of the power of attention and mental concentration, a dazed condition at times; impairment of memory; at times incoherent and rambling in her talk.

Condition of Speech.—Temporary attacks of partial motor aphasia, with the use of wrong words.

Motor Symptoms.—Partial loss of power in the right arm and leg; tongue protruded to the right; a tendency of the head to fall to the right side.

Knee-jerks.—The right very feeble, left active. *Plantar reflex* active on both sides.

Pseudo-localising Symptoms.—Neuralgic pains in the area of distribution of the left great occipital nerve, temporary double facial paralysis (observed by Dr. Foulis), marked difficulty in swallowing; difficulty in articulation. (These symptoms were suggestive of a lesion in the region of the pons Varolii. Dr. Barlow had also thought that the symptoms, when he saw the patient, were indicative of a lesion "far back.")

Subsequent Progress. — Severe left-sided headache, chiefly frontal and occipital; frequent vomiting, difficulty in mastication, difficulty in swallowing, violent coughing (due apparently to "crumbs going the wrong way" and suggestive of paralysis of the larynx); answers questions in whispers only; ultimately complete right-sided hemiplegia and marked motor aphasia.

Death.—March 19, 1885.

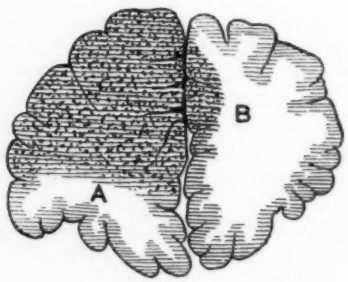

FIG. 5.

Section through the frontal lobes of the brain in the case of A. B. (Case 5), showing the position of the lesion.

The letter *A* points to the lesion in the left frontal lobe.

The letter *B* points to a portion of the right frontal lobe affected by the direct extension of the tumour.

Post-mortem Examination.—No general increase of intracranial pressure, left frontal lobe markedly larger than the right, the dura adherent over the left frontal lobe; a large glio-sarcomatous tumour with a cyst the size of a hen's egg in the left frontal lobe, involving the parts already described in the heading of the case (see fig. 5).

Brain otherwise quite normal.

Remarks. In this case, in which the tumour was situated in the left frontal lobe, the following pseudo-localising symptoms were noted: — Neuralgic pains in the area of distribution of the left great occipital nerve; temporary double facial paralysis (observed by Dr. Foulis); difficulty in mastication; marked difficulty in swallowing; difficulty in articulation (not aphasic in the early stages of the case); coughing, apparently due to paralysis of the larynx ("crumbs getting down the wrong way").

The well-marked mental symptoms, the development, in the later stages of the case, of motor aphasia and of right hemiplegia showed, however, the true position of the lesion.

Case 6 (Case 35 in the Table.) *Large glio-sarcomatous tumour of the left frontal lobe, involving and completely destroying the whole of the first and the anterior two-thirds of the second and third left frontal convolutions; the posterior third of the second and third left frontal convolutions were slightly implicated; the motor area was not involved.*

J. M., aged 52, publican, was seen with Dr. MacGibbon on March 22, 1892.

Duration. Was quite well until three months ago; since then has suffered from headache, mental apathy, loss of memory, drowsiness, flushings of the face, &c.

General Symptoms. Headache (very severe, chiefly frontal); no vomiting; no giddiness; no tenderness on skull percussion; drowsiness; semi-comatose condition; incoherence of speech; answers questions in monosyllables; talks nonsense; involuntary micturition and defæcation; double optic neuritis. Pulse 45; temperature 98°.

Speech. Motor aphasia appears to be present, but this point is difficult to determine owing to the drowsy, apathetic condition of the patient.

Motor Symptoms. None.

Knee-jerks. Not obtained; but I speak with hesitation on this point; the patient was a very big, fat man, lying in bed in a semi-stupid condition, and the knee-jerks were consequently very difficult to examine.

Subsequent Progress. Patient died the day after my visit, in a condition of coma.

Post-mortem Appearances. Great increase of intracranial pressure. Left frontal lobe markedly larger than the right; it contained a large glio-sarcomatous tumour which had destroyed the parts enumerated in the heading (see fig. 6).

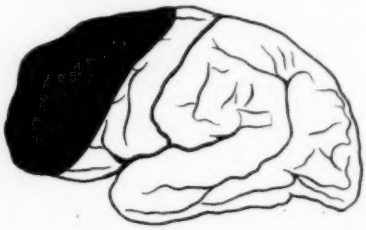

FIG. 6.

Left hemisphere of the brain in the case of J. M. (Case 6), showing the position of the tumour.

Remarks. In this case mental symptoms were very marked; motor aphasia appeared also to be present in the later stages of the case.

(B.)—CASES IN WHICH THE FRONTAL LOBE WAS INVOLVED ALONG WITH OTHER PARTS.

Case 7. (Case 6 in the Table). *Large sarcoma springing from the dura, causing extensive atrophy and destruction of the right ascending frontal and ascending parietal convolutions, the inferior and middle frontal convolutions, the outer half of the island of Reil and the anterior end of the temporo-sphenoidal lobe. No paralysis.*

M. D., aged 20, single, shop-girl, admitted to the Newcastle-on-Tyne Infirmary on January 5, 1877, complaining of headache, vomiting and giddiness. These symptoms thought to be due to "biliousness."

Previous History. Eleven years ago, received a head injury. For the past three years has suffered from headache. Eight months ago, the headache became more severe and the patient began to vomit. Two months ago, she had to give up her situation on account of these symptoms. She has more than once tumbled off her chair, she thinks because of giddiness.

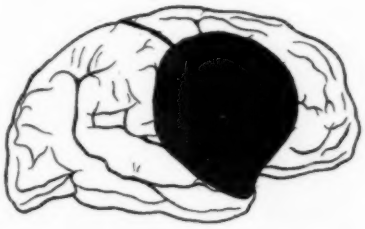

FIG. 7.

Right hemisphere of the brain in the case of M. D. (Case 7), showing the position of the lesion.

General Symptoms. Headache very severe; worst at night, frontal and vertical; vomiting frequent; giddiness frequent; double optic neuritis, very marked in degree.

Mental Symptoms. None except that the patient is much quieter than she used to be.

Speech. No defect.

Motor Symptoms. Absolutely none.

Knee-jerks. Not examined.

Subsequent Progress. Improved under large doses of iodide of potassium.

On *February* 6, 1877, died in an epileptic fit.

Post-mortem Appearances. Bone over the right motor area distinctly thin. The dura mater adherent on the right side over the inferior frontal and inferior parietal areas. Great increase of the intracranial pressure; the convolutions of both hemispheres were much flattened and the sulci effaced. A large sarcomatous tumour, which measured from before backwards 3 inches, from above downwards 3 inches, and from without inwards 1½ inches, sprang from the dura on the right side and

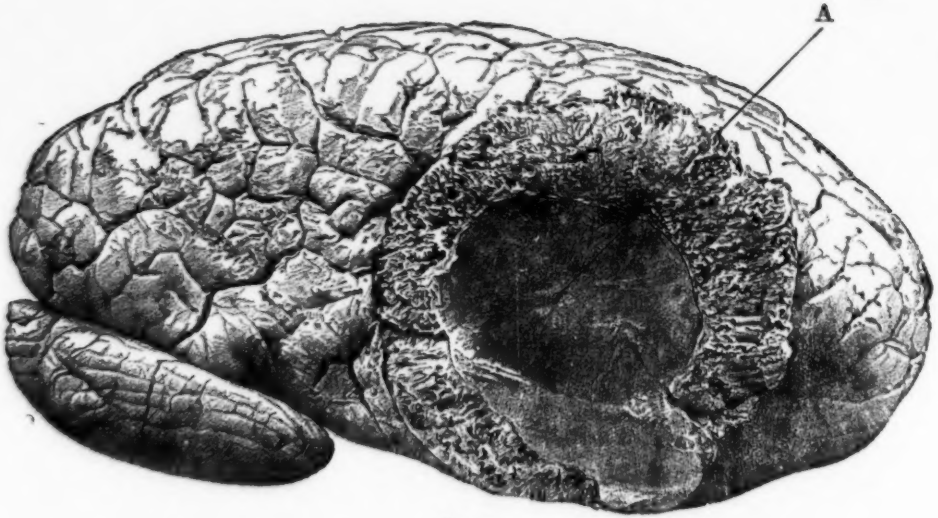

FIG. 8.

The outer surface of the right hemisphere of the brain in the case of M. D. (Case 7), showing the tumour *in situ*. (Copied from a photograph and somewhat reduced in size.)
The letter *A* points to the outer surface of the dura over the centre of the tumour.

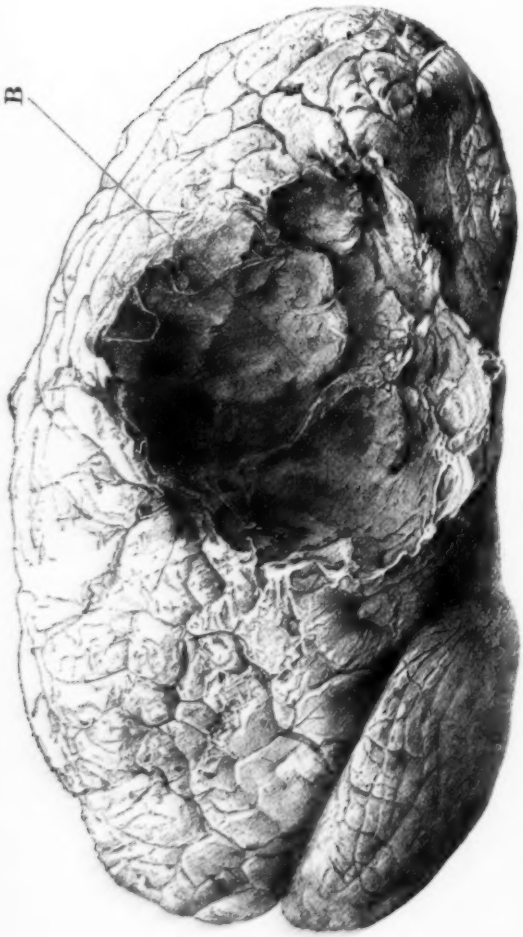

FIG. 9.

The outer surface of the right hemisphere of the brain, in the case of M. D. (Case 7), showing the brain after the tumour was removed. (Copied from a photograph and somewhat reduced in size.)
The letter *B* points to the extensive depression on the surface of the brain into which the tumour fitted.

had destroyed the areas of the cortex enumerated in the heading (see figs. 7, 8, 9 and 10).

The brain and tumour were exhibited in the Pathological Museum of the Edinburgh Meeting, 1898.

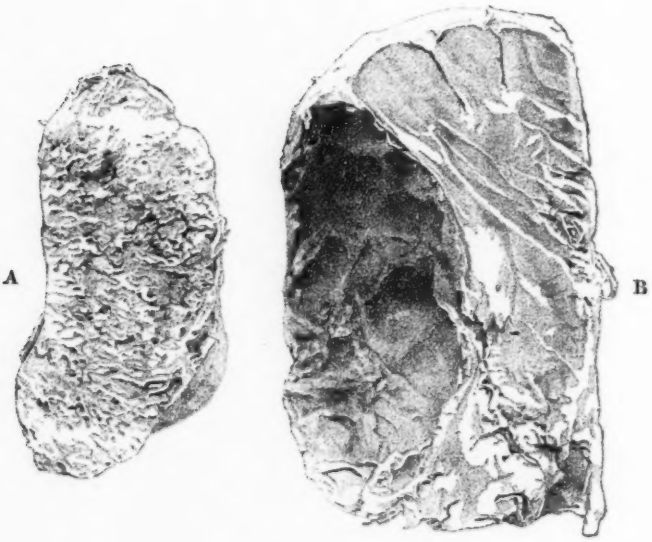

FIG. 10.

Transverse vertical section through the right hemisphere of the brain, in the case of M. D. (Case 7), showing the tumour after section, and the depression in the brain tissues produced by it. (Copied from a photograph and somewhat reduced in size.)

The letter *A* points to the tumour.

The letter *B* is placed on the inner (left) side of the right hemisphere, which has been separated from the opposite (left) hemisphere by a section from before backwards in the middle line.

Remarks. In this case a very large portion of the right motor area (ascending frontal and parietal convolutions) of the adjacent (2nd and 3rd right) frontal convolutions and the anterior end of the right island of Reil were completely destroyed, and yet there were no localising symptoms—absolutely no paralysis.

Case 8. (Case 7 in the Table). *Six tubercular tumours of considerable size in different parts of the cerebral hemisphere:* (1) *which measured* $\frac{3}{4}$ *in. by* $\frac{1}{2}$ *in., situated in the first left frontal convolution;* (2) *which measured* $\frac{3}{4}$ *in. by* $\frac{1}{2}$ *in., in the tip of the left occipital lobe;* (3) *which measured* $1\frac{1}{2}$ *ins. in diameter, in the head of the left corpus striatum;* (4) *which measured* $\frac{5}{8}$ *in. by* $\frac{5}{8}$ *in., in the right angular gyrus;* (5) *which measured* $1\frac{1}{4}$ *ins. by* $\frac{3}{8}$ *in., in the left gyrus fornicatus;* (6) *which was rather larger than the preceding, situated in the right gyrus fornicatus.*

J. W., aged 5, admitted to the Newcastle-on-Tyne Infirmary on February 27, 1877, suffering from headache and vomiting.

Previous History. Patient received a severe injury to his head two years ago. For the past three months has suffered from headache and vomiting.

General Symptoms. Headache (very severe, frontal, usually worse at night); vomiting; double optic neuritis very marked; voracious appetite.

Localising Symptoms. Impairment of sensibility to touch and pain over the greater part of the body. The left wrist-joint, which is in a state of tubercular disease, is not painful even when pressed and moved.

Mental Symptoms. None observed at the time of the patient's admission to hospital; he was a sharp, intelligent boy. Later, he became dull, stupid and then drowsy.

Motor Symptoms. No paralysis except internal strabismus of the left eye.

Knee-jerks not examined.

Subsequent Progress. On July 5, the patient had a series of general epileptiform convulsions in which the left side of the body was chiefly affected. During his stay in hospital, optic atrophy with blindness developed. He became stupid and drowsy.

On *August* 21, 1877, seven months after admission to hospital, he died.

Post-mortem Appearances. Scalp, bones and membranes were natural. Convolutions were much flattened and the sulci effaced. The brain weighed 2 lbs. $14\frac{1}{2}$ ozs. On cutting into the cerebral substance, tubercular nodules were found in the situations enumerated in the heading. A large, dry, caseous mass was present in the lower lobe of the left lung. Some recent grey tubercles throughout the left lung and in the apex of the right lung. The left wrist-joint was in a state of tubercular degeneration.

Remarks. In this case, six tubercular tumours of considerable size were situated in different parts of the cerebrum (*see* heading), and, except that sensibility to touch and pain were markedly impaired (probably as the result of destruction of parts of both gyri fornicati), no definite localising symptoms were observed.

Case 9. (Case 19 in the Table). *Large glioma and cyst in the left hemisphere, causing complete destruction of the greater part of the left ascending frontal and ascending parietal convolutions and the subjacent white matter, the posterior end of the third left frontal convolution and the greater part of the white matter of the left frontal lobe. No motor paralysis till four days before death. No aphasia.*

M. R., aged 29, female, single, right-handed, admitted to the Edinburgh Royal Infirmary on February 6, 1897, suffering from symptoms suggestive of hysteria or a cerebral tumour.

Previous History. As a girl, the patient suffered from attacks of *petit mal*. *Two years ago*, after the sudden death of her mother, she began to suffer from fits, some of which were hysterical and others epileptiform in character. The epileptiform attacks commenced with a tingling in the right hand and right leg. *Eight weeks before admission*, the patient had an attack of influenza, and since this attack has been confined to bed for the greater part of the day. *Three weeks before admission*, she, for the first time, complained of headache; it was very severe and referred to the left side of the head and face; of vomiting and dimness of vision. She was nervous and distinctly hysterical. *Four days before admission to hospital*, she complained of slight weakness in the right hand, but no definite loss of power was noticed until two days before admission. On the day of her admission to hospital it was noticed that there was slight dragging of the right leg.

For the past month, the patient has been in a peculiar mental condition, and was thought both by her friends and medical attendant to be hysterical.

Condition on Admission. Patient was in a stupid, dazed, and apparently hysterical condition. There was slight weakness of the right orbicularis palpebrarum, but no other obvious paralysis. She frequently put her right hand up to her head. She resisted vigorously with the right as well as with the left arm while the optic discs were being examined with the ophthalmoscope. She

spoke very little and chiefly in monosyllables. Double optic neuritis was marked.

Speech. No speech defect except feebleness of articulation had been noticed before her admission to the hospital.

Knee-jerks, plantar and conjunctival reflexes were normal and equal on the two sides.

Diagnosis. An intracranial tumour; situated in the left hemisphere; nature probably gliomatous.

There were no localising symptoms except the facts:—That the headache before admission had been left-sided; that two days before admission to hospital there had been slight weakness of the right arm; that on the day of her admission to hospital there had been slight dragging of the left leg; and that as the result of my examination slight weakness of the right orbicularis palpebrarum muscle was found to be present. I could not satisfy myself that there was any paralysis of the arms or legs.

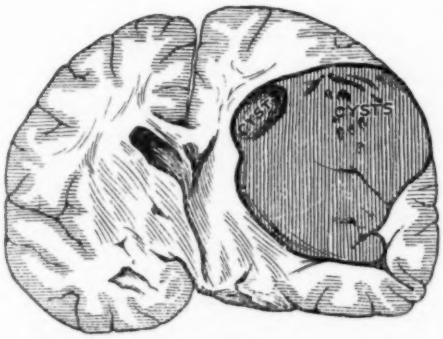

FIG. 11.

Transverse vertical section through the brain in the case of M. R. (Case 9), showing the position and extent of the tumour.
The extent of the tumour is shown by a black line.

Subsequent Progress. The patient died in a convulsion the night after her admission to hospital.

Post-mortem Appearances.—Scalp, skull-cap and membranes normal. Convolutions greatly flattened and sulci effaced, especially over the left hemisphere. The left parietal and frontal lobes considerably larger than the right. A large gliomatous tumour, in which there were several cysts, had infiltrated and destroyed the lower half of the left ascending frontal and parietal

convolutions and the white matter passing down from these convolutions to the internal capsule; the posterior end of the second and third left frontal convolutions and their subjacent white matter; the left island of Reil and the left external capsule (see fig. 11). The lenticular nucleus was infiltrated and displaced downwards and inwards, its grey outline being still recognisable. The grey matter of the left third frontal convolution was not completely destroyed, but the white matter immediately subjacent to it was completely infiltrated and destroyed. The upper half of the motor cortex was not directly affected, but the conducting fibres passing from the leg and upper arm centres to the internal capsule were markedly compressed and displaced inwards, *i.e.*, to the right.

The other parts of the brain were absolutely normal. Sections of the brain, showing the tumour, were exhibited in the Pathological Museum of the British Medical Association at the Edinburgh Meeting, 1898.

Remarks. In this case, in which a very large gliomatous tumour with cystic formation had invaded and completely destroyed the lower half of the left ascending frontal and ascending parietal convolutions and the subjacent white matter, there was practically speaking no paralysis. The greater part of the posterior end of the third left frontal convolution and the whole of its subjacent white matter were infiltrated and destroyed, and yet there was no motor aphasia. The patient was right-handed.

Temporo-Sphenoidal Lobe.

In the next two cases (10 and 11) the tumour involved the temporo-sphenoidal lobe as well as the frontal lobe. In Case 10, the tumour, which apparently had commenced in the left temporo-sphenoidal lobe, had ultimately extended to the left frontal and left occipital lobes. In Case 11, two separate tumours were present—one in the right temporo-sphenoidal and the other in the left frontal lobe.

The first case (Case 10) is of great importance, because the first and second left temporo-sphenoidal convolutions were completely cut off by destruction of their subjacent white matter from the other parts of the brain, and the third left frontal convolution and its subjacent white matter

were entirely destroyed; and yet there was no word-deafness and no motor-vocal aphasia.

I may here state that in another case of tumour which I examined *post mortem* some years ago, but which was not under my own care during life, a large glio-sarcomatous tumour had entirely destroyed the posterior half of the first and second left temporo-sphenoidal convolutions, in a right-handed person, without the production of word-deafness. That patient, who was right-handed, suffered from symptoms which were suggestive of hysteria.

The notes of Cases 10 and 11 are as follows:—

Case 10. (Case 22 in the Table). *Enormous gliomatous tumour involving and destroying the greater part of the left frontal and temporo-sphenoidal lobes, and a large part of the left occipital lobe. No aphasia.*

W. S., aged 32, labourer, was sent to me by Dr. Sym as an out-patient at the Edinburgh Royal Infirmary on December 3, 1896, suffering from double optic neuritis and partial right-sided homonymous hemianopsia.

Previous History. The patient, *who is a right-handed man*, was quite healthy till *two-and-a-half years ago.* He then had an epileptic fit. The epileptic fits have continued on an average once a month since; they are general and are not preceded by an aura or any definite localised commencement. *For the past two-and-half years*, the patient has at times complained of slight headache. *Three months ago*, he fell down in the street, apparently as the result of a fit; he remained insensible for thirteen hours after this attack, and has not worked since. On recovering from this attack he complained of dimness of vision in the left eye and pain over the right eye. Since this attack he has been duller and quieter than he was before; his memory has become impaired and he has been absent-minded. He has frequently complained of giddiness.

Condition on Examination. Marked double optic neuritis; partial right-sided homonymous hemianopsia; slight impairment of the power of convergence.

Speech Functions absolutely normal; no word-deafness, no word-blindness.

Motor Functions. No paralysis except the defective convergence mentioned above. *Knee-jerk* slightly more marked on the right than on the left side.

Diagnosis. An intra-cranial tumour; situation left hemisphere, probably far back in the region of the occipital lobe. The only localising symptoms were partial right-sided homonymous hemianopsia, the slight impairment of the power of convergence, the defective contraction of the pupils on convergence, and the slight increase of the knee-jerk on the right side.

Subsequent progress of the Case. December 30, 1896. The patient is duller mentally. The dimness of vision is more marked. The hemianopsia appears to be *in statu quo*. There is slight paralysis of the right external and left internal recti muscles, and defective convergence of the left eye.

During the next three months, the patient continud to suffer from frontal headache and occasional epileptiform attacks. His mental condition became worse, his memory more impaired. He became duller and more stupid. On several occasions he failed to recognise friends in the street (psychical blindness). His sight became more impaired.

In June 1897, the headache was more severe, frontal. The mental dulness and stupidity were much increased. Sight was worse. The gait was distinctly staggering.

On June 20, 1897, he was admitted into the Edinburgh Royal Infirmary. He was very dull and stupid. Could give little or no information about himself. *Hearing* appeared to be slightly defective in both ears. *Speech*: There was no word-deafness and no motor-vocal aphasia. When asked how old he was, where he was born, what his occupation was, where he lived and where he was at the present time, he was unable to answer correctly; but he answered ordinary questions, which did not involve any effort of memory, at once and quite well; he seemed to have no loss of words. The most careful enquiry failed to elicit any evidence of word-deafness or of motor vocal aphasia. There were, in fact, no aphasic defects (word-blindness, word-deafness, or motor-vocal aphasia) during the whole course of the case. *Sight* was *nil* in the left eye and very slight in the right; consequently, at this stage his power of reading and writing could not be tested. Inability to write appeared chiefly to be due to loss of vision, partly perhaps to the weakness and incoordination of the right hand which were present, and partly to the fact that he could not concentrate his attention and make the mental effort required. *Smell* seemed to be normal in both nostrils. *Sensibility* to touch, pain, heat and cold normal, except that there was slight difficulty in localising tactile impressions correctly over both legs.

Motor Functions. Distinct loss of power in the right side of the face, in the right arm and in the right leg. Grasping power as measured by the dynamometer—right, 70; left, 105.

Reflex Functions. Right knee-jerk increased, left diminished. Cremasteric reflex very active on the left, absent on the right side. Fæces passed in bed, apparently as the result of the mental dulness; has some control over the bladder.

September 5, 1897. Right-sided hemiplegia more marked, Mentally duller. Otherwise *in statu quo.*

It was decided to trephine over the left frontal lobe with the object of relieving the increased intra-cranial pressure. The profound mental symptoms suggested that the tumour involved the frontal lobe; but the partial right-sided homonymous hemianopsia which was present at the commencement of the case was suggestive, as has already been stated, of a lesion in the left occipital lobe. The right-sided hemiplegia showed that the motor area or conducting motor tracts in the left hemisphere were implicated. It was obvious, therefore, if the frontal lobe was involved, either that the tumour was of very great size or that there was more than one tumour.

September 9. Mr. Stiles trephined over the left frontal lobe. Scalp, skull cap and membranes were normal. The intra-cranial pressure was enormously increased. No tumour was found. (*Post-mortem* examination showed that the tumour involved the frontal lobe just below the trephine opening.) The patient recovered satisfactorily from the immediate effects of the operation; a cerebral hernia, however, formed at the seat of the trephine wound. The patient's mental condition did not improve after the operation, but he was able to move his right leg a little better than he could before the operation. A few days after the operation a large quantity of cerebro-spinal fluid soaked through the dressings, and large quantities continued to soak through until the date of the patient's *death, October* 8, 1897. After the operation the patient became very thin and took little food.

Post-mortem Appearances. Trephine wound over the upper part of the left frontal lobe. Hernia cerebri surrounded by localised meningo-encephalitis. Great increase of the intra-cranial pressure. Convolutions flattened, sulci effaced. Left hemisphere of the brain much larger than the right and occupied by an enormous gliomatous tumour, which involved the left frontal, temporo-sphenoidal and occipital lobes. The tumour extended from within a quarter of an inch of the tip of the left frontal lobe right back to a point one inch in front of the tip of

the left occipital lobe. The second and third left frontal convolutions and their subjacent white matter were entirely destroyed. The whole of the white matter of the left temporo-sphenoidal lobe was entirely destroyed. The grey matter of the left temporo-sphenoidal lobe was almost entirely, but not completely, destroyed; the connections of the grey matter of the first and

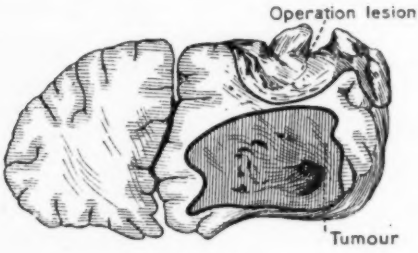

FIG. 12.

Transverse vertical section through the frontal lobes of the brain in the case of W. S. (Case 10), showing the position of the tumour and of the lesion (cerebritis) which resulted from the operation of trephining.

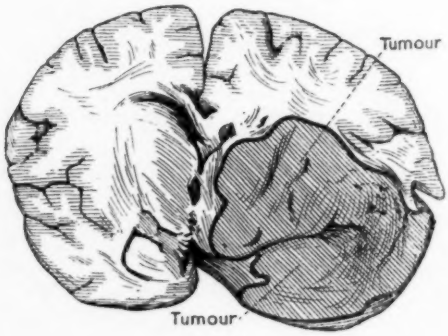

FIG. 13.

Transverse vertical section through the middle of the brain in the case of W. S. (Case 10), showing the extent of the tumour in the temporo-sphenoidal lobe.

The limits of the tumour are represented by a black line.

second left temporo-sphenoidal convolutions were entirely cut off by the tumour in the substance of the lobe (see figs. 12 and 13). A large part of the white matter of the anterior two-thirds of the left occipital lobe was completely destroyed by the new growth.

The parts specified above were not merely infiltrated with gliomatous tissue, but were entirely disorganised and destroyed.

Sections of the brain were exhibited in the Pathological Museum of the Edinburgh Meeting of the British Medical Association, 1898.

Remarks.—In this case, in which the third left frontal convolution and its subjacent white matter were completely infiltrated and destroyed, and in which the greater part of the grey matter of the first and second left temporo-sphenoidal convolutions and the whole of their subjacent white matter were completely infiltrated and destroyed by the new growth, there was no motor-vocal aphasia and no word-deafness.

The extensive destruction of the left temporo-sphenoidal lobe was attended with no localising symptoms, unless the deafness (partial and bilateral) was—and this seems probable—due to this cause.

The loss of memory and the mental impairment were very marked, and suggested a lesion of the frontal lobe.

The partial hemianopsia and the psychical blindness suggested a lesion of the occipital lobe.

Case 11. (Case 37 in the Table). *Glio-sarcomatous cyst in right temporo-sphenoidal lobe, and a gliomatous tumour, with recent hæmorrhage, the size of a large walnut in the posterior end of the second left frontal convolution.*

X. Y., aged 63, excise officer, seen December 23, 1893, complaining of headache, giddiness and dimness of vision.

Previous History. Six months ago the patient was suddenly seized with a sharp pain, as if he had been struck with a stone, behind the right ear. He was quite well previously. He has, at times, suffered from the same pain since; and the headache, giddiness and dimness of vision have since developed.

General Symptoms. Headache (intense, right-sided, deep and internal); also shooting, superficial, neuralgic pains referred to the right side of the head and the lobe of the right ear; no vomiting; giddiness very marked; dimness of vision marked (is unable to read any test types at 18 feet). Double optic neuritis very marked.

Mental Symptoms. Absolutely none.

Speech Symptoms. None.

Motor Symptoms. None.

Knee-jerks equal and normal on the two sides.

Diagnosis. An intracranial tumour, probably in some part of the right hemisphere (because of the locality of the pain). Absolutely no other localising symptoms.

Subsequent progress of the Case. March 20, 1894. Rather better. Neuralgic pain less. Complains of pain, numbness and coldness behind the right ear and down the right side of the neck. Optic neuritis distinctly less; sight somewhat clearer.

In April, 1895, spat up a considerable quantity of bright fluid blood.

May 28, 1895. Gave a somewhat exaggerated statement of the amount of blood which he had spat up. Is in a restless mental condition. Sight greatly improved; says that he is "able to play a fair game at golf." For the past year, has had little or no headache or giddiness. Still complains of a numb, cold feeling behind the right ear. The optic neuritis has quite disappeared, leaving slight neuritic atrophy; his sight is dim, but he has no difficulty in reading and writing (tested by myself).

In April and May, 1896, had three epileptiform attacks.

June 18, 1896. Had another severe fit yesterday. The onset of the attack was preceded by an aura of jargon-reading (he was reading aloud to his daughter at the time and suddenly commenced to read a lot of nonsense and jargon which she could not understand), and by dimness of vision; he lost consciousness; there were no spasms. The attack was followed by complete temporary motor aphasia, word-blindness and agraphia.

On examination, June 18, 1896, I found the patient in a dazed and confused mental condition. He knew Dr. Menzies, but did not know me. There was no word-deafness. There was slight motor aphasia (he used some wrong words); but he answered most questions which were put to him quite correctly. There was distinct word-blindness and some agraphia. He could not read *his own name*; could not read the letter *a*. When asked to write his name, wrote his Christian name correctly and his surname incorrectly, and then read what he had written as his own name. Could not read his name (when written correctly) a few minutes afterwards. There did not appear to be any hemianopsia, but owing to the confused mental condition of the patient it was difficult to determine this point with certainty. There was no paralysis. The knee-jerks were equal and normal.

On August 4, had another fit.

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For some weeks after this attack, the patient improved.

On August 29, was very well; took a drive and walked two miles of the way home.

After this date, he got worse; suffered from headache and fearful giddiness. While in bed during the first half of September, he wrote a few short business communications and read the newspaper. After this date, he did not write at all, was unable to sign his own name (to draw a cheque). His doctor thought that the inability to write was due to dimness of vision, but this is perhaps doubtful. Did not read. At times he became very excited ("pranced round the room in his nightshirt," &c.); at times was mentally bright, at other times seemed almost in a condition of dementia. There was never any motor paralysis; he could hold a pen quite well.

Two days before death he became rapidly worse (semi-comatose); and it was apparently at this date that the recent hæmorrhage, described above, occurred.

He died on *October* 22, 1896.

Post-mortem Appearances. Scalp, skull-cap and dura were normal. There was no meningitis. The vessels were healthy. The convolutions on both sides of the brain, but especially on the left side, were moderately flattened and the sulci to some

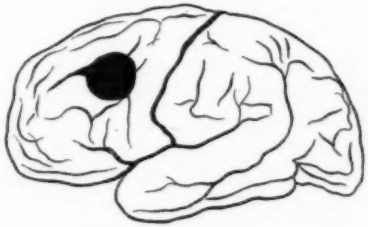

FIG. 14.

Left hemisphere of the brain in the case of X. Y. (Case 11), showing the position of the tumour.

extent effaced. The middle part of the second right temporo-sphenoidal convolution had a yellowish appearance, as seen from the outside, as if from internal softening. In the right temporo-sphenoidal lobe there was a slit-like cavity (cyst) measuring two inches in length by half an inch in breadth at the thickest part. This cavity was surrounded by condensed brain tissue, which on microscopical examination was found to be gliomatous in character.

A recent hæmorrhagic extravasation the size of a large walnut was situated in the posterior end of the second left frontal convolution. On microscopical examination, the hæmorrhage was found to be poured out from the vessels of a gliomatous tumour. The tumour and hæmorrhage were sharply localised to the posterior end of the left frontal convolution (see fig. 14). The third left frontal convolution and the subjacent part of the motor area (left ascending parietal convolution) were not implicated. There was no lesion of the left angular gyrus or supramarginal convolutions. The other parts of the brain were healthy.

The lungs and heart were healthy. No intrathoracic cause for the hæmoptysis was found.

Remarks.—There is every reason, I think, to suppose that in this case the lesion (gliomatous tumour and cyst) in the right temporo-sphenoidal lobe was the first to develop, and that the tumour in the second left frontal convolution was only developed in the later stages of the case. Until shortly before the patient's death, there were absolutely no localising symptoms (unless the pain in the right side of the head and the numbness and coldness behind the right ear were due to the tumour in the right temporo-sphenoidal lobe). The general symptoms of an intracranial tumour (headache, giddiness and double optic neuritis) were, however, marked from the first.

In the later stages of the case, more definite localising symptoms were developed, viz., epileptiform attacks, one of which commenced with jargon-reading and was followed by temporary motor-vocal aphasia, word-blindness and agraphia. During the last six weeks of his life the patient was unable to read and to write; his medical attendant thought this inability was due to loss of vision, but the correctness of this opinion is, perhaps, doubtful. The agraphia was certainly not due to motor paralysis; the patient could hold a pen quite well. The question, of course, arises whether the agraphia was not directly due to the very localised lesion (tumour) in the posterior end of the second left frontal convolution (Exner's writing-speech centre), and whether the word-blindness was not indirectly due to the same lesion. It is, of course, impossible to answer the question, either one way or the other, with certainty; but

the possibility of the agraphia and word-blindness being due to this cause (destruction of Exner's writing-speech centre) must be entertained; and in this connection it must be remembered that in Case 4, in which the posterior end of the second left frontal convolution was completely destroyed by a tumour, and in which there was no agraphia, the destruction was very slow and chronic. Personally, I see no reason to believe in the existence of a special writing-speech centre, but I record the present case, which may perhaps be advanced in support of Exner's view, impartially and with an open mind.

Summary of the Localising Symptoms (in the Cases which have been Related) Suggestive of a Tumour in the Frontal Lobe.

In seven of the eleven cases in which the frontal lobe was involved (viz., in Cases 1, 2, 5, 6, 9, 10, and 11), well marked mental symptoms were present; in the remaining four cases (viz., 3, 4, 7 and 8), there were no definite mental symptoms. Nevertheless, I am strongly of opinion that tumours localised in the frontal lobe are more apt to be attended with well marked mental symptoms (mental enfeeblement, loss of memory, loss of the power of attention, change in disposition, irritability, taciturnity, obstinacy, incoherence and rambling of speech, delusions of suspicion, &c.) than localised tumours in any other part of the cerebral hemispheres.

In Cases 1 and 2, in which the tumour involved identical parts of the frontal lobe (in Case 1 the right and in Case 2 the left frontal lobe being the seat of the tumour), the mental symptoms were practically the same, viz., a marked change in disposition and temper; at times, both patients were irritable and obstinate, at other times, sullen, stupid and depressed; in both cases memory was markedly impaired; and in both cases there was grinding of the teeth during sleep.

In one Case (5) a marked tendency of the head to fall to the opposite side was noted.

Tumours in the posterior end of the frontal lobe frequently (by pressure on, or implication of, the motor fibres passing from the motor cortex to the internal capsule) produce loss of power on the opposite side of the body (Cases 1, 2, 3, 5,); the loss of power sometimes affects both legs, and in some cases the loss of power frequently recurs as a temporary condition—sudden giving way of the leg or legs (Case 1).

Tumours on the surface of the posterior end of the frontal lobe may, by irritation of the adjacent motor cortex, produce Jacksonian epilepsy (Case 3).

Tumours in the posterior end of the left frontal lobe, which involve the motor-speech centre, may produce motor-vocal aphasia (Cases 5 and 7), but the motor-vocal speech centre or its subjacent white matter may be entirely destroyed by a tumour without any motor aphasia (Cases 9 and 10).

In Case 4, complete but slow destruction of the posterior end of the second left frontal convolution was not attended with any agraphia; in Case 11, destruction of the posterior end of the second left frontal convolution was (perhaps) attended with agraphia and some word-blindness.

A voracious appetite was present in one case (8).

The *general symptoms* which result from tumours of the frontal lobe are in no way peculiar.

In the 11 cases detailed above *headache* was present in 10 (and not noted in 1 case); in 7 of the 10 cases the headache was very severe. In 3 cases the headache was frontal; in 2 cases, frontal and vertical; in 1 case, frontal and occipital; in 1 case, frontal and parietal; in 1 case, frontal and temporal; in 1 case, referred to the right side of the head and face (the side of the tumour); and in 2 cases the locality of the headache is not noted.

Vomiting is perhaps less frequent and I think usually less severe than in tumours situated further back. In the 11 cases recorded above, vomiting was present in 6 (marked in 5, slight in 1) and absent in 5 cases.

Giddiness was present in 6 cases (marked in 5, slight in 1); absent in 2 cases, and not noted in 2 cases.

Optic neuritis was present in 9 cases (marked in 7, slight or moderate in 2); in one case the optic neuritis was much more marked in one eye (that on the side of the tumour); absent in 1 case and not noted in 1 case.

General epileptiform convulsions occurred in 6 cases; and localised spasms (Jacksonian epilepsy) in 2 cases; in the remaining 3 cases there were no spasms or convulsions.

Condition of the knee-jerks. In 5 of the 11 cases the condition of the knee-jerks was not noted (these cases all occurred more than twenty years ago); in 3 cases the knee-jerks were equal and normal on the two sides; in 1 case, in which the tumour was situated in the left frontal lobe (Case 5), the knee-jerk was feeble on the right, and active on the left side; in 1 case, in which the tumour involved the left frontal, temporo-sphenoidal and occipital lobes (Case 10), the knee-jerk was increased on the right and diminished on the left side.

Summary of the Localising Symptoms (in the Cases which have been Related) due to a Tumour in the Temporo-sphenoidal Lobe.

In both of the cases (10 and 11) in which the tumour involved the temporo-sphenoidal lobe there were, practically speaking, no localising symptoms; and I have been in the habit of regarding the temporo-sphenoidal lobe as *par excellence the* silent area of the brain. So far as my experience enables me to judge, localising symptoms are most certainly and obviously produced in those cases of intra-cranial tumour in which the tumour is situated at the base of the brain, and in which the cranial nerves at their point of origin from the nervous tissues are directly involved by the new growth. Then, in the relative order with which they can be most easily and certainly diagnosed (*i.e.*, in which the localising symptoms are most certain and constant), I would place tumours in the following situations:—(2) pons Varolii and medulla oblongata; (3) centrum ovale (cases in which the fibres of the internal capsule or the optic radiations of Gratiolet are directly implicated)

(4) tumours of the occipital lobe, involving the half-vision centre or the optic radiations of Gratiolet; (5) tumours of the motor area, in which the tumour produces irritation and gives rise to localised epileptiform convulsions; (6) tumours of the cerebellum; (7) tumours of the frontal lobe; (8) tumours of the upper part of the parietal lobe (*i.e.*, tumours which do not involve the angular gyrus or its subjacent white matter); and last of all, the most difficult to diagnose and locate, (9) tumours of the temporo-sphenoidal-lobe, and especially, of course, tumours of the right temporo-sphenoidal lobe.

I need hardly say that this is merely a rough classification. Tumours of the *central* lobe of the cerebellum, for instance, can usually be located and diagnosed with great certainty. The same statement probably also applies to tumours involving the left angular gyrus (in right-handed people).

In Case 10, there was some impairment of hearing in both ears. In Case 11, the patient complained of pain, numbness and coldness behind the right ear (the side of the tumour); this was perhaps a localising symptom. Tumours of the left temporo-sphenoidal lobe may of course give rise to word-deafness, but this symptom was not present in Case 10, in which the left temporo-sphenoidal lobe was, practically speaking, completely destroyed.

Island of Reil.

In addition to Cases 9 and 10, in which the tumour involved the left island of Reil, I have met with one other case in which the island of Reil (on the right side) was destroyed by a tumour. The notes are as follows:—

Case 12. (Case 18 in the Table). *Two gliomatous tumours—(1) involving the left centrum ovale, and (2) the right island of Reil, lenticular nucleus and adjacent parts.*

M. P., aged 63, female, nurse, a right-handed woman, admitted to the Edinburgh Royal Infirmary on September 25, 1896, suffering from loss of power in the right arm and leg, numbness in the right side of the body, headache, giddiness and occasional dimness of vision.

Previous History. Has suffered from headache for five weeks. Numbness and loss of power in the right arm commenced three weeks ago; it first affected the shoulder and gradually extended down the arm; and then affected the leg.

General Symptoms. Headache; occasional giddiness; optic neuritis present (verified on *post-mortem* examination).

Motor Symptoms. Complete loss of power in the right arm, partial loss of power in the right leg. No paralysis of the right side of the face.

Knee-jerks both exaggerated.

Sensibility. Some impairment of tactile sensibility on the right side of the body (arm and leg). Sight good. No hemianopsia.

Speech. The articulation a little thick, but no aphasic defect.

On *September* 28, 1896, patient took what she termed "a funny turn," shivered but did not feel cold; there was no rise in temperature; she felt in a nervous peculiar state. During the attack, twitchings were observed in the upper part of the right arm, the right side of the chest and the right side of the abdomen. There was no loss of consciousness. The nurse said that she had difficulty in speaking during the seizure. The attack was not followed by any increase of the paralysis. Sight was dim for two hours after the attack. At 8 *p.m.*, she complained of severe headache, frontal, affecting both sides. At 9.30 *p.m.*, she vomited and there was distinct difficulty of articulation and in expressing herself. At 11 *p.m.*, she was stupid and seemed to have difficulty in understanding what was said to her, and in answering questions, but there was no defect of articulation.

October 12, 1896. Did not answer the House-Physician at his evening visit.

October 13, 1896. Has a distressed look; cannot be got to answer any questions; complete motor aphasia; appears to understand everything that is said to her and makes signs intelligently in answer to the questions which are put to her, but does not speak. The *left* side of the face is paralysed.

October 16, 1896. The paralysis of the left side of the face is more marked. Temperature 101°.

October 18, 1896. Looks rather better. Answered some questions to-day intelligently, but for the most part in monosyllables. In answer to the remark, "Glad you are better," answered, "Yes"; in answer to the question, "Have you any pain?" answered, "No"; in answer to the question "Is your arm feeling any stronger?" answered "A little better."

October 19, 1896. The left side of the body became rigid, and the patient passed into a condition of coma. Both arms appear to be paralysed. Eyeballs while at rest turned to the right; nystagmoid jerkings of the eyeballs to the left, with slow return to the middle line, were present; pupils equal and dilated; they react to light; conjunctival reflex present. Pulse and respirations both very rapid. Some difficulty in swallowing.

October 23, 1896. Patient died. The temperature ran up to a high point just before death. For eighteen hours before death, the respirations numbered 70 per minute.

Post-mortem Appearances. Scalp, skull-cap and membranes flattened and sulci effaced. Weight of the brain = 3 lbs. 4 ozs. Arteries at the base normal. On cutting up the brain, a glio-sarcomatous tumour, measuring $1\frac{3}{4}$ in. by 1 in. in breadth, was present in the white matter of the left centrum ovale, just in front of the left angular gyrus.

In the right hemisphere, a recent hæmorrhage which subsequent microscopic examination showed had been poured out from the vessels of a gliomatous tumour, had almost completely destroyed the lenticular nucleus and the anterior part of the right internal capsule. In front of the hæmorrhage a mass of gliomatous tissue involved the caudate nucleus and the greater part of the island of Reil and its subjacent white matter. There were numerous small hæmorrhages in the gliomatous tissue which infiltrated the island of Reil.

Remarks. The most important point in this case, so far as the present discussion is concerned, is that a tumour with hæmorrhage in the *right* island of Reil and adjacent parts produced motor aphasia, which would no doubt, if the patient had lived, have been merely temporary. I have met with other cases in which a sudden lesion (hæmorrhage, &c.) in the region of the *right* motor-vocal speech centre has in right-handed persons produced temporary motor-vocal aphasia.

Occipital Lobe.

In my experience tumours are comparatively rarely met with in the occipital lobe. Two cases only, verified by *post-mortem* examination (in addition to Cases 8 and 10, which have already been detailed), have been under my own observation during life. The notes are as follows:—

Case 13. (Case 11 in the Table). *Sensory (visual) Jacksonion epilepsy, due to the presence of a cicatrix, the result of an old syphilitic gumma on the outer surface of the posterior end of the left occipital lobe.*

W. G., aged 60, usher in a school, formerly a soldier, seen at the Edinburgh Royal Infirmary on March 5, 1887, complaining of frequent flashes of red and white light in the right eye.

Previous History. At the age of 24, the patient had syphilis (a serpiginous scar on the tibia, the result of an old syphilitic ulceration, is still present). The same year (1848) he received a severe head injury. *In* 1863, he complained of severe headache limited to the back and top of the left side of the head; the pain was worse at night; at this time a swelling developed $1\frac{1}{2}$ inches behind and slightly above the left parietal eminence, and he took an epileptic fit. He has had several epileptic fits since; the third and fourth, which occurred during the years 1883 and 1884, were preceded by flashes of light in the right eye. *From* 1877 *up to* 1887, when I first saw the patient, he continued at long intervals to have flashes of light in the right eye. The attacks occurred on an average about once a year; each attack, which consisted of very numerous paroxysms, usually lasted for three or four weeks. The paroxysms occurred every five or ten minutes, and lasted from half a minute to three minutes; they consisted of flashes of light in the right eye; the light was said to be red and white like a magic lantern. For some days after the attack the patient was unable to see to the right side (right-sided homonymous hemianopsia).

Present Condition. When the patient came under my observation he was suffering at frequent intervals from the paroxysms described above. The fields of vision were carefully mapped out with the perimeter (the charts have been published in my "Atlas of Clinical Medicine," plate xxxvi., figs. 3 and 4). They show right-sided homonymous hemianopsia both for white and colours, and some contraction of the sound (seeing) halves of the visual fields. During the paroxysms, the patient complained of a noise like a steam-engine in the left ear. The fundi were quite normal and there was no word-blindness.

Progress of the Case. Under treatment (iodide of potassium) the paroxysms and flashings soon subsided and the hemianopsia almost completely disappeared, the fields for white and green remaining somewhat contracted (see charts, "Atlas of Clinical Medicine," plate xxxvi., figs. 5 and 6). The headache also completely disappeared. The patient was at the time suffering

from an epithelioma of the tongue, and operative procedure (which would otherwise have been considered) was contra-indicated.

The patient died several months after he first came under my observation.

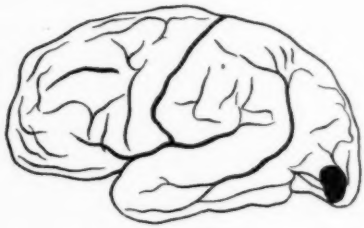

FIG. 15.

Left hemisphere of the brain in the case of W. G. (Case 13), showing the position of the lesion.

Post-mortem Appearances. The membranes were adherent over the outer surface of the anterior part of the left occipital lobe and over the adjacent surface of the cerebellum (see fig. 15, also "Atlas of Clinical Medicine," plate xxxvii., fig. 4). The brain substance was sclerosed and atrophied and had evidently at one time been the seat of inflammatory changes (syphilitic gumma, &c.).

Remarks.—In this case an irritative lesion (cicatrix of an old syphilitic gumma) on the outer surface of the posterior end of the left occipital lobe produced frequently recurring attacks of sensory (visual) Jacksonian epilepsy—flashes of light referred to the right eye—followed by temporary sensory paralysis (*i.e.*, right-sided homonymous hemianopsia).

Case 14. (Case 32 in the Table). *Numerous melanotic sarcomatous tumours in the brain, one situated in the tip of the left occipital lobe, another in the middle lobe of the cerebellum, which was entirely destroyed.*

S. E. M., aged 26, female, single, seen on September 28, 1886.

Previous History. Patient has had a pigmented mole on the right shoulder all her life. After a direct injury (blow with a

stick) received about the age of 12 or 13, the pigmented mole enlarged. In 1881, it was the size of a half-crown piece; it was then scraped. In 1885, it had returned and then formed a fungating tumour the size of a walnut. On October 23, 1885, it was excised. The patient remained well until July 26, 1886, when she began to complain of flashes of light in the eyes, intense headache and vomiting.

Condition on September 28, 1886. *General Symptoms.* Headache (intense, especially on the right side); vomiting; giddiness; double optic neuritis.

Localising Symptoms. Patient suffered very frequently (50 or more in the twenty-four hours) from paroxysmal attacks lasting for five minutes at a time, from headache, flushing and flashes of light in the eyes; the light was said to be especially bright in the right eye; it had the brilliancy of an electric light, and varied in

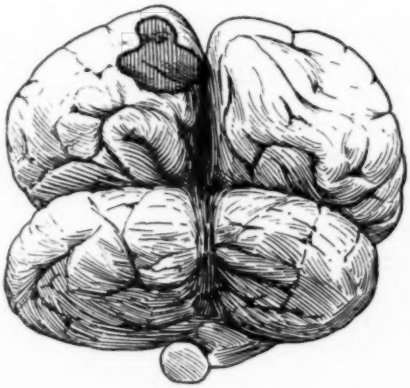

FIG. 16.

Posterior end of the brain in the case of S. E. M. (Case 14), showing the position of the lesion (outlined by a black line).

size from a button to a large globe. During these paroxysms the patient stated that she was completely blind. After an attack, which I personally witnessed, passed off, there did not appear, so far as could be determined by the rough test, to be any hemianopsia.

Mental Faculties. Quite clear. No emotional or hysterical symptoms.

Motor Symptoms. Difficulty in directing the movements of the left arm. *Knee-jerks* active and equal.

Diagnosis. Melanotic sarcoma in the brain; probably several deposits, one involving and irritating the half vision centre in the left hemisphere.

Subsequent Progress. The patient continued to suffer from the same symptoms. During the month of October, 1886, paralysis of the left arm and leg developed.

On *November* 2, 1886, the patient died.

Post-mortem Appearances. A nodule of melanotic sarcoma, the size of a large walnut on the surface of the posterior end of the left occipital lobe just above its tip (see fig. 16). The subjacent white matter was infiltrated, but only partly destroyed. Numerous other melanotic deposits in the brain, the frontal and occipital lobes being chiefly involved. A small nodule in each island of Reil. A nodule of some size in each optic thalamus. A small nodule in the pons involving the motor tract on the right side. A large nodule in the middle lobe of the cerebellum, which was entirely destroyed. Several small nodules in the lateral lobes of the cerebellum.

Remarks.—In this case, irritation of the left half-vision centre produced frequently recurring flashes of light in the eyes, especially in the right (opposite) eye.

In addition to the two cases which have just been related, the occipital lobe was also the seat of a tumour in two of the cases which have been already described (Cases 8 and 10). In Case 10, there was partial right-sided hemianopsia and mind-blindness due to involvement of, either the white matter of the anterior end of the left occipital lobe, below the angular gyrus, or of the parietal lobe.

Parietal Lobe.

Only one case with *post-mortem* has come under my observation in which the angular gyrus was involved by a tumour. The notes of that case, in which the right angular gyrus and the adjacent parts were the seat of the lesion, have little value for the present discussion. The case

occurred twenty-four years ago. My want of knowledge, at that time, of the localising symptoms due to brain lesions and the defective mental condition of the patient sufficiently account for the comparatively imperfect nature of the clinical record. It is briefly as follows:

Case 15. (Case 1 in the Table). *Syphilitic tumour involving the right angular gyrus and the adjacent parts of the parietal and occipital lobe, with extensive softening of the subjacent white matter.*

R. S., aged 41, chemist, single, admitted to the Newcastle-on-Tyne Infirmary on November 12, 1874, suffering from left-sided hemiplegia, headache, &c.

Previous History. The patient had syphilis many years ago. Has suffered from headache and "rheumatism" for the past year. Three months ago left-sided hemiplegia suddenly developed.

General Symptoms. Headache (marked) mental impairment and loss of memory; left hemiplegia and left hemianæsthesia affecting the arm and leg; dimness of vision; post-neuritic atrophy, and syphilitic choroiditis; tibial nodes. The patient was fat and well nourished.

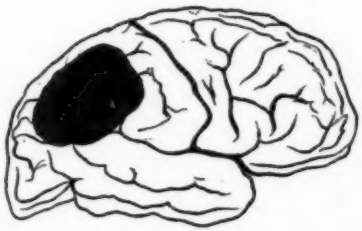

FIG. 17.

Right hemisphere of the brain in the case of R. S. (Case 15), showing the position of the lesion.

Subsequent Progress. The patient continued to suffer from recurring headaches. There was some slight improvement in the hemiplegia and hemianæsthesia.

On December 8, 1874, he had a general epileptic fit which was followed by coma and death.

Post-mortem Appearances. A hard, nodulated, old syphilitic tumour, the size of a walnut, was situated on the outer surface of the right parietal lobe. The dura over the posterior end of the right hemisphere was thickened. The angular gyrus, the upper and middle parietal convolutions, the anterior end of the occipital convolutions and the subjacent white matter of the *right* hemisphere were implicated and more or less extensively destroyed by softening (see fig. 17). Syphilitic cicatrices were present in the liver and nodes on the tibia.

Remarks.—With reference to the occurrence of word-blindness in cases of cerebral tumour, I may again point out that in Case 11 there was distinct but temporary word-blindness, apparently due to the (indirect) irritation produced by a tumour which was sharply limited to the posterior end of the second left frontal convolution.

Case 16. (Case 39 in the Table). *Fibro-cellular tumour involving the left upper parietal convolution, entirely latent, complicated with a large cerebral abscess, acutely developed in the subjacent white matter.*

A. B., aged 40, seen on December 4, 1895.

Previous History. Patient was a strong, healthy man who had never been previously ill. He had not suffered from syphilis or ear disease, and he had never received any head injury. On December 2, 1895, he complained of pain in the left parietal region. On December 3, he experienced a numb feeling in the right hand and some difficulty in writing and using his knife at lunch. Thinking these symptoms were due to biliousness, he took a blue pill before going to bed on the evening of December 3. During the night of December 3 he had three very severe epileptic fits; the convulsions were general and attended with loss of consciousness. After the fits he complained of great pain in the abdomen.

Present Condition. When seen by me on the morning of December 4, the patient was quite sensible and able to give a detailed account of his sensations, &c. The pulse and temperature were normal; there was no paralysis; the knee-jerks were equal and normal; the pupils were equal and active; the optic discs were normal; there was no hemianopsia and no aphasia, either sensory or motor; there was no tenderness on skull percussion; hearing was normal, and there was no ear disease.

The chief complaint was abdominal pain and tenderness, which, after careful consideration, it was decided were the result of the violent spasmodic contractions of the abdominal muscles during the fits. The urine was normal; the heart and arteries were normal. There was no suspicion of alcoholic excess or of any form of intoxication.

On December 9, the abdominal pain and tenderness had disappeared. There had been no return of the convulsions. The temperature had remained subnormal; the pulse rather slow (60 to 65 per minute). Since the last consultation there had been more or less headache, and at times it had been very severe; it was chiefly referred to the left side of the head. For the past two nights the patient had been very restless and anxious about himself, and had complained of a numb feeling in the right arm and leg.

On examination, distinct loss of power in the right side (face, arm, and leg) was found to be present. The loss of power was more marked in the leg than in the arm. There was also some loss of tactile sensibility on the right side of the body, more particularly in the leg. There was no aphasia, no word-deafness, no word-blindness, no motor vocal aphasia, and no hemianopsia. Well-marked double optic neuritis was now present. The pupils were equal and active; temperature normal; pulse 60; tongue clean. The urine still continued free from albumin. The heart and arteries were perfectly normal.

On December 11, the patient, who had had a restless night, was obviously worse, the paralysis much more marked. There was also now some difficulty in speaking, apparently due to motor aphasia. Impairment of tactile sensibility on the right side of the body was also more marked. The optic neuritis was more distinct. The pupils still remained equal, and contracted actively to light. The temperature was 92·2°, and the pulse 64. Since the date of the last consultation two days ago, there have been no convulsive twitchings and no severe headache.

On the afternoon of this day, December 11, Dr. John Duncan met Drs. Russell Wood, Playfair, and myself in consultation. He agreed as to the difficult and exceptional nature of the case, and as to the probability of the lesion being thrombosis rather than tumour. With us he thought that in the absence of any obvious cause, cerebral abscess was a most unlikely condition, and might in all probability be excluded. Under these circumstances it was decided not to advise any operative procedure.

On December 12, the patient was worse; motor aphasia

more marked; temperature normal; pulse 60; the condition otherwise unchanged.

At 1.30 a.m. on December 13, the breathing became stertorous, and the patient passed into a condition of coma, the temperature running up in the course of a short time to 102·6°. Death occurred at 4 a.m.

At the post-mortem examination, which was made by Professor Robert Muir, the convolutions of the brain were found to be flattened and effaced, more especially on the left side. At one or two places on the surface of each hemisphere the subarachnoid fluid was slightly turbid, and there were minute particles of lymph in the sulci. The membranes at the base were quite healthy. There were no emboli or thrombi in any of the vessels. The arteries and veins of the brain were absolutely normal.

On examining the surface of the brain, a tumour was found on the left side. Its posterior border was situated at the junction of the parietal and occipital lobes, and its inner border half an inch from the middle line. It was irregularly quadrangular in shape, about three-quarters of an inch in diameter, and extended down into the brain as a blunt wedge for about three-quarters of an inch. The tumour was sharply marked off from the surrounding brain tissue.

On transverse section through the brain, an abscess the size of a large hen's egg was found in the middle of the left hemisphere. It was situated in the centrum ovale, rather nearer the posterior than the anterior end of the brain. At its nearest point to the cortex it was about an inch below the grey matter. The contents of the abscess consisted of a thin and extremely stinking pus. The brain substance forming the wall of the abscess was fairly firm and dense, more especially at one point underlying the tumour.

The dura mater, the venous sinuses, the bones of the skull, the middle and internal ear, the nose, etc., were all carefully examined, and were found to be absolutely normal. The heart, arteries, lungs, liver, spleen, kidneys, and other abdominal and pelvic viscera were quite healthy. The most careful search failed to detect any local source for the abscess, or any disease in any of the organs of the body other than the brain.

(The full notes of the case, with a discussion of the diagnosis and a detailed account of the treatment which was adopted, are published elsewhere.[1])

[1] *Scottish Medical and Surgical Journal*, Nov., 1897, p. 972.

Basal Ganglia.

(*a*) *Optic Thalamus.*

In one case (Case 13 in the Table), the tumour seemed to have its primary seat in the right optic thalamus, but there were no symptoms to indicate that this was the position of the lesion. The case was perhaps the most deceptive, so far as localising symptoms were concerned, which has come under my notice. The notes are as follows:—

Case 17. (Case 13 in the Table). *Glio-sarcoma of the right optic thalamus; symptoms indicative of a syphilitic gumma involving the right leg centre.*

R. G., aged 50, an old soldier, gatekeeper at Holyrood, was seen at the Edinburgh Royal Infirmary on March 18, 1893, suffering from headache, numbness and stiffness in the left leg, occasional spasmodic twitchings in the left leg beginning in the left big toe.

Previous History. Had syphilis 26 years ago.

General Symptoms. Headache (severe, referred to the right motor area, worse at night); tenderness on pressure and percussion over the right motor area, exactly localised to the leg and upper arm centres; giddiness (occasional); the patient had on more than one occasion tumbled, he thought partly as the result of giddiness and partly in consequence of giving way of the left leg; the discs were ill-defined, the veins in the right fundus being dilated, but there was no definite optic neuritis.

Localising Symptoms. Tenderness over the leg and arm centres on the right side; attacks of Jacksonian epilepsy occurring several times a day, the spasm beginning in the left big toe; exaggeration of the left knee-jerk. There was no swelling or alteration in the scalp at the seat of the pain and tenderness.

Diagnosis. A cortical gumma in the position of the right leg centre.

Treatment. Iodide of potassium, twenty grains three times daily.

Subsequent Progress of the Case. Speedy relief of the headache and disappearance of the spasms. When last seen, the patient said that he was quite well, but some tenderness on percussion could still be elicited over the right motor area. Was advised to continue the iodide.

Some weeks later, the head symptoms returned and were not relieved by the continued administration of the iodide. The patient was therefore sent by his medical attendant (who knew that I had no male beds) into the Infirmary, where he was seen by Dr. Gibson, who, not knowing that I was connected with the case, arrived at the same diagnosis—a cortical gumma in the right-leg centre—and advised, as the iodide had failed to give relief, that the patient should be trephined. This was accordingly done by Mr. Alexis Thomson. No tumour was found. The operation wound healed satisfactorily. The patient died some time afterwards.

Post-mortem Appearances. The scalp, skull-cap and membranes quite normal. Some flattening of the convolutions and obliteration of the sulci, especially on the right side. A glio-sarcomatous tumour about the size of a small egg occupied the position of the right optic thalamus and had slightly encroached upon the motor fibres of the pyramidal tract.

Remarks. In this case, the symptoms were clearly indicative of a syphilitic gumma of the right leg-centre; the tumour was a glioma of the right optic thalamus; it had apparently produced irritation of the leg fibres of the internal capsule.

In addition to this case, in which the optic thalamus was the primary seat of the new growth, the optic thalamus was involved along with other parts in three other cases (10, 13, and 23). In Case 13, which has already been related, melanotic deposits of considerable size were present in each optic thalamus. In Case 23 in the Table, a very large glio-sarcomatous tumour, which had commenced in the right centrum ovale and involved the whole of the internal capsule and the optic radiations of Gratiolet, had extended to the right optic thalamus; but, since tumours of the centrum ovale are not included in this discussion and since there were no symptoms suggestive of involvement of the optic thalamus, I do not propose to describe this case in detail.[1] In Case 10, the left optic thalamus was also invaded by the tumour.

[1] I hope at some future period to publish reports of my other cases of intra-cranial tumour.

In none of these four cases in which the optic thalamus was invaded by a new growth were there, so far as I could detect, any definite symptoms due to the lesion in this area of grey matter, unless the paroxysms of flushings, which were very marked in Case 13, were due to this cause.

(*b*) *Lenticular Nucleus.*

In two cases (Case 12, which has already been detailed, and Case 24, which will afterwards be described), the lenticular nucleus was completely destroyed by a new growth. In Case 12, the motor fibres of the adjacent pyramidal tract and the island of Reil were also involved and destroyed by an acutely developed lesion. In that case there were well marked symptoms of motor paralysis on the opposite side of the body, and motor aphasia, but no symptoms indicative of a lesion of the lenticular nucleus. In Case 24, in which the left lenticular nucleus was completely replaced and destroyed by a deposit of secondary cancer, there were absolutely no symptoms indicative of the presence of a lesion in this part of the cerebral tissue.

(*c*) *Caudate Nucleus.*

In two cases (8 and 12) the caudate nucleus was invaded by a new growth. In Case 8, which has already been detailed, six scrofulous tumours were found in various parts of the cerebral tissue. The tumour which was present in the head of the left corpus striatum and which had entirely destroyed this portion of grey matter, did not appear to give rise to any definite symptoms. In Case 12, which has also been described, the caudate nucleus was involved along with the lenticular nucleus, the island of Reil, and the motor strands of the internal capsule. In that case the involvement of the adjacent parts produced well marked symptoms—hemiplegia on the opposite side, and motor aphasia, followed by coma. Consequently, distinctive localising symptoms due to the involvement of the head of the corpus striatum could not be expected to have been noticed.

Cerebellum.

In 7 cases the cerebellum was alone involved by the new growth. In 3 other cases the cerebellum, as well as other parts, was the seat of a tumour.

The following are the details of the cases:—

Case 18. (Case 4 in the Table). *Subtentorial sarcoma springing from the membranes and deeply indenting the left lateral lobe of the cerebellum; unsuspected during life.*

A. C., aged 54, female, admitted to the Newcastle-on-Tyne Infirmary on September 28, 1875, suffering from mitral stenosis, cardiac dropsy, right-sided hemiplegia (marked) of three weeks' duration.

General Symptoms. Headache; vomiting; no optic neuritis, but veins of the fundus large and tortuous, the right disc much redder than the left. Patient was in a stupid mental condition and could hardly be got to answer questions. During her stay in hospital she suffered from trembling fits, in which she was not cold.

Localising Symptoms. With the exception of the hemiplegia there were no localising symptoms—nothing to suggest a lesion of the cerebellum. It was, however, noted that when the patient attempted to stand she tumbled over, partly apparently from loss of motor power, partly from loss of control. The patient professed not to hear skull-conducted sounds in the left ear, and neither aërial nor skull-conducted sounds with the right ear.

Result. On October 21, 1875, patient died suddenly in a convulsion, in which the right side of the body appeared to be more affected than the left.

Post-mortem Appearances. A round tumour the size of a greengage plum was found below the tentorium cerebelli on the right side. It sprang from the dura and had deeply indented the lateral lobe of the cerebellum. No signs of softening or inflammation of the cerebellar tissues. A small hæmorrhage, evidently of some weeks' duration, in the extra-ventricular portion of the corpus striatum.

Marked mitral stenosis.

Remarks. In this case, so far as was ascertained, there were absolutely no symptoms suggestive of an intracranial tumour.

Case 19. (Case 8 in the Table). *Large scrofulous tumour in the right lateral lobe of the cerebellum.*

A. C., aged 9, female, admitted to the Newcastle-on-Tyne Infirmary on January 24, 1878, suffering from headache, blindness and deafness.

Previous History. Four months previously the patient had fallen and received a severe blow on the back of the head; the injury was followed by headache and vomiting. A week before admission the patient suddenly lost both sight and hearing.

General Symptoms. Headache (very severe, frontal, especially on the right side of the head). The headache was so intense that the patient, before admission to the Infirmary used to hold her head under the cold-water tap in order to relieve it. Vomiting. Double optic neuritis (marked). Giddiness not complained of.

Mental Symptoms. None. The patient, though blind and deaf, is quite bright and intelligent; she is able to distinguish her mother from other persons by the feel of her dress.

Motor Symptoms. None. The gait is natural except for the defect produced by loss of sight. No paralysis.

Knee-jerks not tested.

Sight. Absolutely blind.

Hearing. Totally deaf both to aërial and skull sounds in both ears.

Subsequent progress of the Case. Patient remained in the Infirmary until June 23, 1878, when death took place.

During her stay in hospital, she suffered from periodic attacks of severe headache and vomiting. She remained absolutely blind and deaf. She had recurring convulsions in which there was no loss of consciousness. The spasms were tonic in character and affected the head and neck, the eye-balls (which were turned upwards) and both upper extremities. Clonic spasms also occasionally occurred in the right side of the face. During the last month of her life she became stupid and apathetic, and both the contents of the bladder and rectum were evacuated involuntarily.

Post-mortem Appearances. The scalp was natural, the skull-cap very thin; it showed no signs of the former injury. The membranes were normal, the convolutions much flattened and the sulci effaced. The dura was adherent over the right lateral lobe of the cerebellum. The right lateral lobe of the cerebellum was considerably enlarged. The increase in size was due to the presence of a scrofulous tumour in its interior. The inner half

of the lobe was chiefly invaded by the new growth. The other lobes and the peduncles were healthy. The ventricles were dilated. The brain substance was œdematous and soft, but otherwise normal. The internal ears and auditory nerves seemed quite healthy to the naked eye.

The bronchial and mesenteric glands were enlarged and caseous. Several deposits of miliary tubercles were present in the lungs and kidneys.

Remarks. In this case, the patient suddenly became absolutely blind and completely deaf, the intelligence and mental activity remaining quite bright.

Case 20. (Case 15 in the Table). *Four tumours in the cerebellum —three in the left, and one in the right, lateral lobe; no symptoms suggestive of cerebellar disease.*

C. McN., aged 18, seen at the Edinburgh Royal Infirmary on October 24, 1896, complaining of headache, vomiting and emaciation.

Previous History. In June of the present year, the elbow joint was excised for tubercular disease. In July the patient commenced to suffer from headache.

General Symptoms. Headache (very severe, frontal and vertical, sometimes passing through to the back of the head, increased by movement); vomiting (very frequent, incessant for the past week, occurring especially in the morning, brought on by movement); marked double optic neuritis; great emaciation; general motor weakness; marked constipation; great dilatation of the pupils.

Localising Symptoms. Slight weakness of the right hand; dynamometer, right=70, left=80. Knee-jerks equal and not exaggerated. Absolutely nothing else. No giddiness. No peculiar gait, though patient is weak on his legs. No spasms. No hemianopsia. No disturbance of sensation. No speech defects.

Mental Symptoms. The patient is inclined to be drowsy, but is intelligent. There is no impairment of memory. The patient was sent to Mr. Cotterill's wards, with a view to operation.

Subsequent Progress. Intense headache; incessant vomiting; nothing would remain on the stomach, and he had to be fed by nutrient enemata. As no treatment appeared to give relief, it was decided to trephine with the object of relieving the increased intracranial pressure.

The operation was performed by Mr. Cotterill, to whom I am indebted for the following notes of the subsequent progress of the case, on November 11, 1896. The patient was trephined over the left parietal region, the only localising symptom being slight weakness in the right hand. The intracranial pressure was greatly increased; immediately the dura was opened, the brain not only bulged through the opening, but ruptured itself. No tumour was found. A trocar was inserted in various directions into the brain, but no fluid was obtained; it was consequently concluded that the lateral ventricle was not dilated. The operation was followed for a few days by relief; the headache and vomiting were lessened, but the loss of power in the right arm increased and slight loss of power developed in the left leg. After a few days, severe headache and vomiting returned. After consultation a second operation was consequently decided upon, but great doubts were entertained as to the position at which a second trephine opening should be made. There were absolutely no mental symptoms; this seemed to exclude a lesion of the frontal lobe. There was no hemianopsia; this seemed to exclude a lesion of the occipital lobe. There was no word-deafness; this was opposed to a lesion in the left temporo-sphenoidal lobe. The fact that no fluid was obtained when a trocar was inserted deep down in the direction of the left lateral ventricle seemed to show that the ventricles were not dilated, and consequently suggested that the increased intracranial pressure was not due to a subtentorial tumour—a tumour in the cerebellum. It was, therefore, decided, by this process of exclusion, to trephine over the right temporo-sphenoidal lobe. This was accordingly done, but nothing was found. The intracranial pressure on the right side was very markedly less than on the left. The original trephine wound was again opened up. There was marked bulging of the brain at the seat of the first operation (left parietal region). A large trocar was inserted deep down into the brain and a quantity of clear fluid escaped, evidently from the left lateral ventricle. A drainage tube was introduced.

Result of Second Operation. November 25, 1896 (the day following the operation). Patient much brighter, much more sensible and intelligent, answering questions readily and correctly. Sight apparently all right. Distinct word-blindness and right-sided homonymous hemianopsia.

After this date the mental condition continued to improve, the optic neuritis became much less. The word-blindness almost completely passed off, the patient being able to read fairly well,

but he continued absolutely unable to write, even to sign his name. This difficulty was not accounted for by the slight motor paralysis which was present in the right arm; it appeared to be due to destruction of fibres passing from the visual speech-centre to the motor writing-centre. The emaciation continued to increase. The second operation wound healed most satisfactorily.

January 11, 1897. The patient is very much worse. Extremely emaciated. Mentally very dull and apathetic. Answers "Aye" or "Yes" to all questions which are put to him. The scalp is markedly bulging at the seat of the original trephine wound (left parietal region).

February 7, 1897. The patient became noisy and excited. Temperature moderately elevated.

February 19. Patient died.

Post-mortem Appearances. Scalp wounds soundly healed. Bulging of the scalp in the left parietal region. A hernia cerebri projected through the trephine hole in the skull. Trephine wound on the right side of the brain normal and no bulging. Intracranial pressure greatly increased. Convolutions flattened and sulci effaced, especially over the left hemisphere. Four

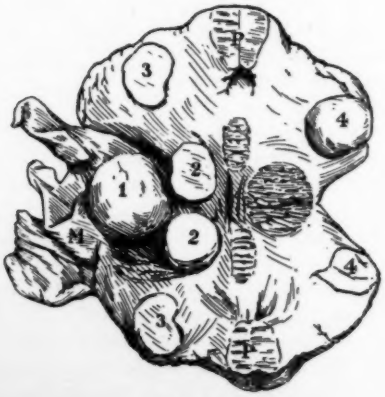

FIG. 18.

Section through the cerebellum and pons in the case of C. McN. (Case 20), showing the position of the tumours.

The letters *P P* are placed on the pons; the letter *M* on a portion of the cerebral membranes.

The figure *1* indicates tumour No. 1, which has not been divided. The figures *2, 2* are placed on tumour 2, *3, 3* on tumour 3, *4, 4* on tumour 4, which have been transversely divided.

scrofulous tumours present in the cerebellum, three in the left and one in the right lateral lobe, in the following situations:—(1) a large tumour, more or less rounded in shape, 1½ ins. by 1¼ ins. in diameter, adhering to the dura, occupying the under-surface and the back of the left lateral lobe of the cerebellum; it had caused extensive destruction both of the grey and the white matter of the cerebellum; (2) a somewhat oval tumour, measuring 1 in. by $\frac{10}{16}$ in. in diameter, situated between the first tumour and the middle lobe; it was situated in the white matter, and was in immediate contact with the large tumour (No. 1) on the one hand and with the middle lobe on the other; (3) a small kidney-shaped tumour measuring $\frac{14}{16}$ by $\frac{14}{16}$ ins. in diameter, in the anterior part of the right lateral lobe; it reached the surface and involved both the grey and white matter of the cerebellum; (4) a tumour, measuring 1 in. by ¾ ins. in diameter, in the lateral part of the right lobe; it reached the surface and involved both the grey and the white matter of the cerebellum (see fig. 18).

Remarks. This was a very remarkable case; for although there were four tumours of very considerable size in the cerebellum, there were no symptoms suggestive of cerebellar disease, except the very severe vomiting.

Case 21. (Case 24 in the Table). *Glioma and cyst of the cerebellum, involving the middle and right lateral lobes.*

A. M., aged 24, a builder, seen May 14, 1885.

Previous History. Four years ago (1880), was seized with causeless vomiting, followed by general torpor and malaise, a reeling gait, but no giddiness. One year ago (1884), suffered from severe headache, chiefly frontal, and referred to the right side of the occiput. During the present year (1885), has continued to suffer from headache and a reeling gait, but does not reel in any particular direction.

Condition on May 14, 1885. Patient complains of constant headache (frontal and right side of the occiput); is apathetic and drowsy; has an inordinate appetite; there is marked double optic neuritis; the urine contains large quantities of phosphates.

Diagnosis. A cerebellar tumour, probably gliomatous in character.

Subsequent progress of the Case. June, 1885. Continues to suffer from headache. Optic neuritis is passing into optic atrophy. Has a frequent, nervous cough. Has had several

attacks of Cheyne-Stoke's respiration. Expression is vacant and staring.

Between June, 1885, *and November*, 1885, continued to suffer from headache. Had several epileptiform attacks in which the right arm and leg were sometimes the only parts which were affected. Giddiness. Temporary œdema of the eyelids. Tinnitus in the right ear and a tendency to fall backwards.

On November 3, 1885, the patient died suddenly after an attack of vomiting.

Post-mortem Appearances. Scalp, skull-cap, membranes and dura natural. Intracranial pressure greatly increased. Convolutions flattened, sulci effaced. A gliomatous tumour, in which there was a large cyst fully the size of a hen's egg, was present in the middle lobe of the cerebellum. The gliomatous tissue involved the right lateral lobe, which was extensively destroyed and atrophied. There was slight atrophy of the left lateral lobe. The pons Varolii and medulla oblongata were markedly compressed and flattened.

Case 22. (Case 27 in the Table). *Glioma of the left lateral and middle lobes of the cerebellum.*

J. J., aged 24, railway surveyor, seen May 12, 1888, suffering from headache, giddiness, vomiting, reeling gait, &c., duration 6 months. The symptoms have been much worse since the patient's marriage four months ago.

General Symptoms. Headache, very severe, chiefly frontal, at times occipital; it is of a beating character. Vomiting, frequent; usually occurs with the headache in the morning before breakfast. Occasional giddiness, especially felt if he walks fast or far. Double optic neuritis, most marked in the left eye.

Localising Symptoms. Tendency to fall to the right side. Impairment of taste in the anterior part of the tongue on the right side. Numbness on the right side of the face. Diminished tactile sensibility on the right side of the face and tongue. Knee-jerks equal and normal.

Diagnosis. A cerebellar tumour involving the central and probably the right lateral lobes; probably a glioma.

Subsequent progress of the Case. Patient did not improve under full doses of iodide of potassium. When next seen (September 6, 1888) headache and vomiting were still frequent and severe, the giddiness and reeling gait more marked, the

optic neuritis more advanced. After this date he rapidly got worse, and died in November, 1888.

Post-mortem Appearances. Scalp, skull-cap and membranes normal. Convolutions much flattened and sulci effaced. A gliomatous tumour, the size of a hen's egg, was present in the cerebellum; it involved the middle and the left lateral lobes. The pons Varolii and medulla oblongata were markedly compressed and flattened.

Remarks. In this case, I expected to find the right lateral lobe involved because of the anæsthesia on the right side of the face and the loss of taste on the anterior part of the right side of the tongue.

Case 23. (Case 29 in the Table). *Gliomatous tumour of the middle and right lateral lobes of the cerebellum.*

A. B., aged 17, seen December 6, 1888, suffering from headache and vomiting, etc. Duration, one year. Supposed cause, a fall from her horse, but this was very doubtful.

General Symptoms. Headache (very severe, chiefly occipital, sometimes frontal); vomiting (frequent, usually occurring with headache first thing in the morning); giddiness (frequent, especially before breakfast before getting up); double optic neuritis (advanced in the left eye to optic atrophy). Anæmia; amenorrhœa.

Localising Symptoms. A staggering and tottering gait; a tendency to fall to the left side; complete loss of vision in the left eye, probably the result of the optic atrophy; vision in the right eye normal; knee-jerks both exaggerated, but especially the right.

No other symptoms.

Subsequent Progress. Under iodide of potassium the patient improved remarkably and, except for the blindness in the left eye, was practically well—able to walk, drive and enjoy herself, read with the right eye, etc.—during the next eighteen months. The headache and vomiting were speedily relieved, the anæmia disappeared, the giddiness became much less marked, and the gait much steadier; the menstruation returned and was regular for several months.

In *June*, 1890, the menstruation ceased, the headache returned, but there was no return of the vomiting or giddiness. The gait

again became worse. Bowels became much constipated. The patient complained of general weakness and shortness of breath.

August 3, 1890. Looks remarkably well in the face; no anæmia. Complains of pain in the back of the head in the centre of the occiput. Tongue furred; bad taste in the mouth. Gait very staggering. Still tends to fall to the left side. Knee-jerks exaggerated on both sides, especially the right. Plantar reflex absent on both sides. Sight in the left eye *nil;* in the right considerably impaired. Optic atrophy present in both eyes; very advanced in the left.

In *November*, 1890, became worse. Headache, chiefly affecting the back and right side of the head. Vomiting and giddiness returned. Difficulty in swallowing developed; fluids regurgitated through the nose. Pulse remained slow, about 60.

On *December* 4, 1890, the vision became suddenly blurred; patient complained of headache; hiccough developed, and she died within the course of a few minutes.

Post-mortem Appearances. Convolutions much flattened and the sulci effaced. Lateral ventricles considerably distended. A gliomatous tumour the size of a small egg was present in the middle lobe and the adjacent part of the right lobe of the cerebellum. The medulla oblongata and pons Varolii were flattened by pressure.

Case 24. (Case 31 in the Table). *Large cancerous tumour in the right lateral lobe of the cerebellum; left lenticular nucleus completely destroyed by a round cancerous nodule; small cancerous nodule in the right ascending parietal convolution.*

M. A., aged 61, seen July 13, 1886, complaining of headache, giddiness, &c.

Previous History. Eight months ago, the left breast and axillary glands were removed for cancer. Three months ago, the patient began to complain of headache, followed by vertigo and a reeling gait. Six weeks ago she had a fit, after which there was temporary paralysis of the right side of the face. One month ago, she had a second fit or fainting, followed by marked excitement which lasted for a week and was succeeded by vomiting.

General Symptoms. Headache (persistent and very severe); vomiting (frequent); vertigo (marked); optic neuritis (intense).

Localising Symptoms. Marked vertigo. Patient says she feels as if the ceiling would fall down on her when her eyes are

open. She says that objects go round her, but cannot tell in what direction they move. No paralysis. No other localising symptoms.

Subsequent Progress of the Case. Headache and other symptoms continued, but no further symptoms developed. There was absolutely no paralysis during the whole course of her illness. For the last month of life the headache, vomiting and giddiness abated, the mental faculties became clouded, and the patient ultimately passed into a semi-comatose condition.

November 25, 1886. Patient died.

Post-mortem Appearances. Increased intra-cranial pressure. Flattening of the convolutions; effacement of the sulci. Ventricular distension. The dura mater was adherent over the right lobe of the cerebellum, which was considerably enlarged and the seat of a cancerous tumour about the size of a hen's egg. The middle peduncle of the cerebellum was considerably compressed.

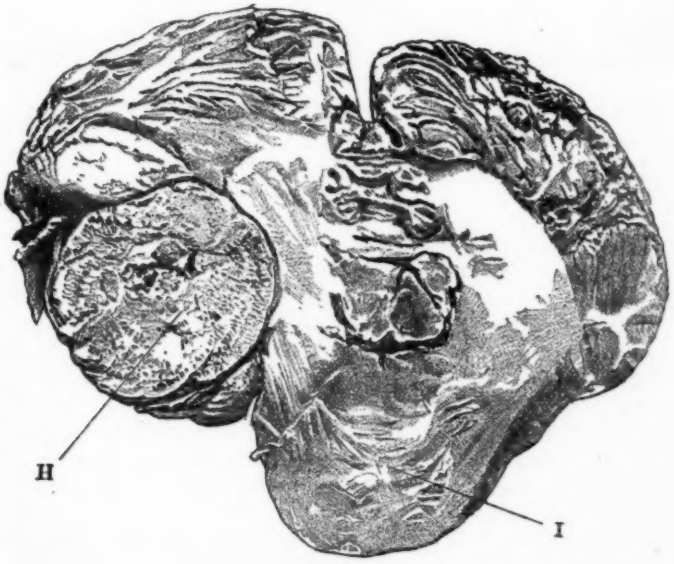

FIG. 19.

Transverse section through the cerebellum and pons Varolii in the case of M. A. (Case 24), showing a large nodule of secondary cancer in the right lateral lobe of the cerebellum.

The letter *H* points to the tumour; *I* to the pons Varolii.

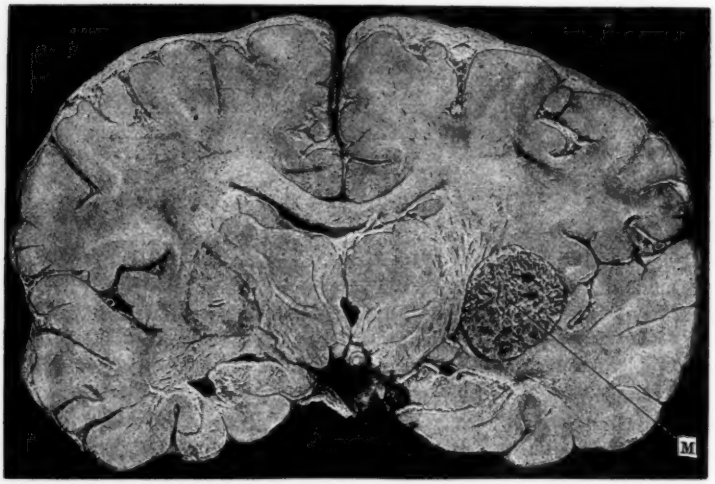

FIG. 20.

Transverse vertical section through the brain in the case of M. A. (Case 24), showing a cancerous nodule (*M*) in the position of the left lenticular nucleus, which was completely destroyed by the new growth.

A large nodule of cancer about the size of a greengage plum occupied the position of, and had entirely destroyed, the left lenticular nucleus (see figs. 19 and 20). A small nodule about the size of a cherry was situated in the upper end of the right ascending partietal convolution. The other parts of the brain were normal.

In three other cases (13, 25 and 26) the cerebellum was also involved by a tumour either springing from the membranes or a tumour which involved other parts of the nervous tissues as well as the cerebellum.

In Case 13, which has already been detailed, a large melanotic deposit had destroyed the central lobe of the cerebellum. There were several other melanotic deposits of small size in both lateral lobes. In this case the vertigo was a striking symptom.

The notes of Cases 25 and 26 are as follows :

Case 25. (Case 10 in the Table). *Scrofulous tumour of the left crus and cerebellum with slight tubercular meningitis.*

J. C., male, aged 1½, seen at the Edinburgh Royal Infirmary on January 24, 1890, suffering from paralysis of the right arm and leg, and paralysis of the muscles supplied by the third nerve on the left side.

General Symptoms. Little or no headache. Vomiting. No optic neuritis. Fontanelle open.

Localising Symptoms. Paralysis of the muscles supplied by the left third nerve, paralysis of the right arm and leg, and rigidity with volitional tremor in the right arm and leg.

Subsequent Progress. Under treatment, the paralysis disappeared. The child got fat, and at the end of nine months was apparently quite well. Ten months afterwards, there was a return of the vomiting, development of headache and symptoms indicative of tubercular meningitis.

The child was admitted to the Sick Children's Hospital, where he died.

Post-mortem Examination. A scrofulous tumour was found at the base of the brain in the region of the left crus cerebri ; another scrofulous tumour in the cerebellum ; and slight recent tubercular meningitis. I have no note of the exact position of the tumour in the cerebellum.

Remarks. The presence of a tumour in the cerebellum was not suspected during life.

Case 26. (Case 40 in the Table). *Glioma of the pons Varolii, crus cerebri and cerebellum.*

A. B., female, aged 7½, seen June 30, 1897, suffering from paralysis of the left arm and leg and paralysis of the muscles supplied by the right third nerve.

Previous History. Patient had influenza three months ago, but seemed to recover perfectly. Three weeks ago, she became more emotional than she used to be, without any obvious cause. Ten days ago, it was noticed that she had some difficulty in moving the left leg ; this gradually increased. The left arm and the left side of the face gradually became affected, and some difficulty in swallowing developed.

General Symptoms. No headache when first seen ; slight afterwards. Vomiting frequent. Commencing double optic neuritis. Constipation. Temperature normal.

Localising Symptoms. Paralysis of the left arm and leg and very slight of the left side of the face. Inco-ordination and volitional tremor on movement of the left arm and left leg. Ptosis of the right eyelid. Paralysis of the right external rectus muscle. Difficulty in swallowing. Knee-jerks absent.

Mental Symptoms. None. The patient is bright and intelligent.

Diagnosis. A scrofulous tumour of the right crus.

Subsequent Progress. Got rapidly worse, much thinner. Occasionally complained of slight headache. The paralysis increased. The vomiting became more frequent, the difficulty in swallowing more marked.

The patient died towards the end of August.

Post-mortem Examination. A tumour of some size was found at the base of the brain on the right side; it involved the anterior part of the right lateral lobe of the cerebellum, the right peduncle of the cerebellum, the right side of the pons, and extended up as high as the right crus. Its limits were not well defined. It was of a dull pinkish colour, of soft consistency, and on microscopical examination was found to be a glioma (the age and family history had suggested tubercle).

In addition to these cases, in which the diagnosis was verified *post-mortem*, I may detail the notes of a case in which all the characteristic symptoms of a cerebellar tumour were present, but in which operative procedure, first over one lateral lobe and then over the opposite lateral lobe of the cerebellum, failed to reveal the presence of a tumour. The notes are as follow:—

Case 27. *Typical symptoms of a cerebellar tumour; operation; no tumour found.*

S. M., female, single, 25, a servant, was admitted to the Edinburgh Royal Infirmary on January 18, 1897, suffering from headache, giddness, staggering gait and loss of vision.

Previous History. The patient was quite well until June, 1896, when the present symptoms (giddiness, difficulty in walking and headache) developed. Some months previously the menstruation had been arrested. In October, 1896, the sight became affected and the patient suffered from vomiting.

Present Condition. Well nourished; no anæmia. Temperature 97·4°

General Symptoms. Headache (severe, occipital, frontal and vertical, especially marked over the right eye and over the left side of the occiput); vomiting (frequent); giddiness (marked). Very marked double optic neuritis, passing into optic atrophy in the right eye.

Localising Symptoms. A staggering gait, with a tendency to fall sometimes to the right, sometimes to the left side, but especially to the left; this was confirmed on several occasions by independent observers. The right leg is thrown out in an incoordinate manner. Knee jerk exaggerated on the right side. No ankle clonus. Plantar reflex absent on both sides. Grasping power in the left hand, as measured by the dynamometer, 55, in the right 70. Romberg's symptom present; tends to fall to the left side when the eyes are closed. Vision in the right eye *nil;* in the left markedly diminished, $= \frac{1}{5}$ (dimness of vision no doubt due to the optic neuritis and atrophy). Nystagmus to the right and upwards; subsequently became marked when the patient looked to the left. The associated movements of the left external and right internal recti muscles weaker than the corresponding movements on the opposite side. Deaf in the left ear both to aërial and bone-conducted sounds; external ear normal. No skull tenderness. No disease of ear. No scalp swelling. Giddiness occurred in paroxysms two or three times a day and lasted from five to ten minutes at a time. Sense of smell completely absent (cause doubtful).

Mentally somewhat dull—a natural defect. Complains every now and again of a cramp-like sensation in the right side of the face.

Diagnosis. A cerebellar tumour, apparently situated in the left lateral lobe; probably a gliomatous cyst.

Subsequent Progress of the Case. Headache continued to recur and to be very severe; it was very definitely located to the left side of the occiput and over the right eye. Vomiting recurred occasionally.

On *February* 8, 1897, the patient was trephined by Mr. Cotterill over the left lateral lobe of the cerebellum. No tumour was found. The intracranial pressure was much increased. Subsequently, a second operation was performed over the right lateral lobe of the cerebellum, but no tumour was found. A good deal of bulging occurred at the site of the operation, especially on the left side. After the second operation the right side of the face and head became markedly swollen.

March 3, 1897. Patient looking very well; much brighter

than before the operation. Headache almost gone. Swelling, scalp and eye, better. Has not vomited since the operation.

She remained in hospital until March 24, 1897, when she was removed by her friends, and died at home a few weeks afterwards.

No *post-mortem examination.*

Remarks. This case illustrates the great uncertainty which there is in the diagnosis of many cases of cerebellar tumour. I am still disposed to think that in it a cerebellar tumour was present, probably situated in the middle lobe and out of reach of the surgeon. The symptoms during life seemed undoubtedly to indicate a tumour in the left lateral lobe.

In Case 22, already detailed, the same difficulty of diagnosis occurred. The symptoms seemed to indicate a tumour in the right lateral lobe, whereas the *post-mortem* showed that the tumour was situated in the left lateral lobe.

But the difficulty of diagnosing cerebellar tumours is not merely confined to the uncertainty of determining in which lateral lobe the tumour is situated. In some cases in which a tumour is situated in the cerebellum, there are no cerebellar symptoms. Case 20, which has already been detailed, in which four tumours were situated in the cerebellum—three in the left and one in the right lateral lobe—is the most striking illustration which has come under my own notice. In that case there were absolutely no localising symptoms to show that the cerebellum was the seat of the lesions.

Cases of Distended Ventricles simulating a Cerebral or Cerebellar Tumour.

Further, in some cases in which the symptoms during life seem clearly to indicate the presence of a cerebral or cerebellar tumour, *post-mortem* examination shows that there is no tumour, but merely a dilated condition of the ventricular system of the brain, more especially of the

fourth ventricle, the result of previous meningitis and obliteration of the foramen of Magendie. The following cases illustrate this point:—

Case 1. *Typical and characteristic symptoms of a scrofulous tumour in the cerebellum; no tumour found post-mortem, merely a dilated condition of the ventricles of the brain and old basilar meningitis.*

M. R., aged 9½, female, admitted to the Edinburgh Royal Infirmary on October 30, 1894, suffering from headache, vomiting, double optic neuritis and paralysis of all four limbs.

Previous History. Five years ago, the patient suffered from tubercular glands in the neck. In August 1893 (fourteen months ago), the present illness commenced without obvious cause. The patient suffered from severe headache, referred to the occiput and to the right temple, and vomiting. There was no fever. Thirteen months ago, her gait became reeling and she tended to fall forwards. One year ago, she became unable to walk; and on more than one occasion, when sitting on a stool, fell backwards, apparently because she overbalanced herself or became giddy. During the summer of 1894, she had an epileptic fit.

Condition on Admission. Is well nourished. Head large; forehead narrow; occiput broad; circumference of head, 21⅝ inches. Head inclined forwards. Rythmical jerking movements, both nodding and rotatory in character, of the head, on voluntary movement. Headache frequent but much less severe than in the earlier stages of the case. Vomiting occasional, every three or four days. Marked double optic neuritis passing into optic atrophy. Vision in the right eye, $\frac{6}{24}$; left, $\frac{6}{18}$. Some internal strabismus of the left eye. Nystagmoid movements of the eyes both on looking to the right and to the left. Pupils dilated and sluggish in their reaction to light. Paralysis of all four limbs, rather more marked on the left side. Legs rigid. Knee-jerks both markedly exaggerated and ankle clonus present in both legs. Volitional tremor and incoördination when patient attempts to make movements with the arms; more marked in the left than in the right arm. Some loss of control over the bladder and rectum. Memory somewhat impaired. Sensibility of the skin, hearing, taste, smell and the muscular sense normal.

Diagnosis. A scrofulous tumour of the cerebellum, probably affecting the middle and left lateral lobes, and pressing on the pons, with secondary hydrocephalus.

Subsequent Progress of the Case. Under drug treatment there was no improvement.

On *December* 24, patient was trephined by Mr. Cotterill over the left lobe of the cerebellum. The intracranial tension was greatly increased. No tumour was found. The headache and other symptoms were relieved for a time. Subsequently, the question of a second operation on the opposite right lateral lobe was considered, but deferred, as the patient's condition did not seem to warrant its performance.

On *January* 14, 1894, the patient died.

At the *Post-mortem Examination* the intra-cranial pressure was found to be greatly increased; the convolutions were flattened and the sulci effaced. A cerebellar hernia had formed at the seat of the trephined wound, and there was slight recent purulent meningitis in the immediate neighbourhood of the wound. The membranes at the base were thickened as the result of old meningitis. The foramen of Magendie was closed. The ventricular system of the brain was very greatly dilated. At the junction of the left crus cerebri with the optic thalamus, a small fibro-caseous nodule about the size of a small pea, in which there were some calcareous gritty particles, evidently an old tubercular lesion, was found.

Remarks. The symptoms in this case (good nutrition, headache, vomiting, giddiness, marked double optic neuritis, absence of fever, a staggering gait, a large head, paralysis of all four limbs, increase of the knee-jerks, jerking movements in the head and limbs on volitional effort, internal strabismus of the left eye and nystagmoid movements on looking *both* to the right and left) were typically characteristic of an intracranial tumour and seemed clearly to show that the tumour was situated in the cerebellum. Further, it should be noted that the head symptoms had commenced without any indications of meningitis, and were of long (fourteen months) duration. The case is a very remarkable one.

Only one other point need be emphasised, viz., the fact that paralysis of all four limbs, with volitional tremor, a spastic condition and exaggeration of the knee-jerks and ankle clonus, seemed undoubtedly to be due to the pressure of the dilated fourth ventricle upon the subjacent pons and medulla.

Case 2. *All the typical symptoms of a cerebellar tumour, found after death to be the result of a dilated condition of the ventricles of the brain and especially of the fourth ventricle, due to obliteration of the foramen of Magendie.*

J. M., aged 19, male, was seen at the Edinburgh Royal Infirmary on May 16, 1888, suffering from headache, vomiting, giddiness, staggering gait, &c.

Previous History. In May 1887, the patient received a head injury; he fell down several feet on to his mouth and was insensible for some hours afterwards. After recovery from this illness, he remained well until three months ago, when the present symptoms developed.

Family History. Two brothers and sisters suffered from "water in the head" and loss of eyesight.

General Symptoms. Headache (frequent and severe); vomiting; giddiness (marked); marked double optic neuritis; staggering gait (patient has on several occasions been thought to be drunk in the street); a tendency to fall backwards in the attacks of vertigo. The patient says that objects go round him from left to right. The patient is well nourished; there is no fever, and no paralysis.

Diagnosis. A cerebellar tumour, probably affecting the middle lobe, probably scrofulous or gliomatous in character.

The patient was admitted to one of the wards of the Infirmary, and died a fortnight afterwards.

Post-mortem Appearances. No cerebellar tumour. Enormous dilatation of the ventricular system of the brain; especially of the fourth ventricle, which formed a large space, the roof of which was bounded by the middle lobe of the cerebellum. The ventricular dilatation was due to thickening and adhesions of the membranes, and closure of the foramen of Magendie. There were no tubercles.

Two other cases of the same nature, verified by *post-mortem* examination, have also come under my observation; but in both of them the previous history was suggestive that the cerebral symptoms, at their commencement, were due to meningitis. I need not, therefore, detail them in full.

The cases which have just been related seem to show that in some cases it is absolutely impossible to differentiate between a cerebellar tumour and a dilated condition of the

fourth and other ventricles due to blocking of the foramen of Magendie. In both of these cases, the cerebral symptoms developed without any obvious cause, and were not preceded by any symptoms indicative of meningitis. In both, the general symptoms of an intra-cranial tumour (headache, vomiting, double optic neuritis and giddiness) were present; and in both, the localising symptoms (marked vertigo and a reeling gait, etc.) were indicative of a cerebellar lesion. In Case 28, there was, in addition, paralysis of all four limbs, with rigidity, exaggeration of the reflexes and volitional tremor, such as one gets in some cases of cerebellar tumour either as the result of the lesion in the cerebellum itself, or the pressure of the cerebellar tumour on the subjacent pyramidal tracts in the pons or medulla.

I have been so much impressed with this uncertainty in diagnosis that, in some of the cases in which the symptoms of a cerebral or cerebellar tumour are present and in which the symptoms subside and the patients get well, I am disposed to think that the lesion was perhaps a dilated condition of the ventricular system of the brain, the result of obliteration of the foramen of Magendie, rather than a cerebral or cerebellar tumour which had become quiescent or had become cured under treatment. Four cases of this kind (apparent cure of a non-syphilitic cerebral tumour) have come under my own notice. In such cases, where the symptoms subside and the patient remains well, it is impossible, I think, in the present position of our knowledge, provided that there were no symptoms suggestive of meningitis in the early stages of the case, to say with certainty whether the case was one of dilated ventricles, the result of obliteration of the foramen of Magendie, or of a cerebellar tumour (scrofulous or gliomatous) which had become quiescent or healed (absorbed). That a gliomatous tumour of the cerebellum may become quiescent is shown by Case 29, which has already been detailed.[1]

[1] In the remarks which I made at the discussion on the Treatment of Intracranial Tumours, which was introduced in the Neurological Section of the British Medical Association by Professor Ferrier, I have referred to this point in more detail (see *British Medical Journal*, vol. ii., 1898, p. 964).

AN INVESTIGATION UPON THE PLANTAR REFLEX, WITH REFERENCE TO THE SIGNIFICANCE OF ITS VARIATIONS UNDER PATHOLOGICAL CONDITIONS, INCLUDING AN ENQUIRY INTO THE ÆTIOLOGY OF ACQUIRED PES CAVUS.

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An important and very definite variation in the plantar reflex occurring in certain pathological conditions, and having considerable clinical value, has been brought into notice by M. J. Babinski.[1] He has found that in certain cases the flexion of the toes upon the metatarsus, which is the initial movement in the normal plantar reflex, is replaced by extension of the toes upon the metatarsus. This phenomenon, which he names "le phénomène des orteils," has been found by him only in those cases where there have been lesions of the pyramidal system as evidenced clinically or by *post-mortem* investigation. An exception must be made in the case of newly-born infants, in which the plantar reflex commences with extension of the toes. Babinski explains this by the fact that in very young children the pyramidal system is incompletely developed. He claims for this sign that it never occurs in normal adults, nor in functional disease, and that of the objective signs of lesions of the pyramidal system it is the earliest to appear after the lesion, and may be the only sign present as in cases of recent hemiplegia, when the myotatic irritability is absent, or diminished; and in combined posterior

and lateral sclerosis with absent knee-jerks and in Friedreichs' disease.

Van Gehuchten[2] has published an investigation of a number of cases, and has confirmed Babinski's results. My attention was first directed to the investigation of variations in the plantar reflex in January, 1898, by Sir William Gowers, who drew my attention to a case of right hemiplegia in which stimulation of the sole of the foot on the hemiplegic side produced invariably a slow extension of the great toe upon the metatarsus and no movement of the other toes or of any other muscle. The plantar reflex on the non-paralysed side was brisk and commenced with flexion of the toes.

The clinical part of this investigation is based upon :—

(1) A hundred adults examined in the medical wards of a general hospital, who being free from acute disease and presenting no sign of any lesion of the nervous system were taken as normal.

(2) A hundred children examined in the wards and out-patient department of the Hospital for Sick Children at Great Ormond Street.

(3) Three hundred cases of nervous disease that have come under my care as House Physician at the National Hospital during the last six months.

I have to thank my chiefs at the National Hospital, Dr. Hughlings Jackson, Dr. Bastian, Dr. Buzzard, Sir William Gowers, Dr. Ferrier, Dr. Beevor and Dr. Tooth for their kind permission to make use of cases under their care in the Hospital. I am also much indebted to Dr. Lees and Dr. Sydney Phillips for permission to examine the patients in their wards at St. Mary's Hospital; to Dr. Lees and Dr. Colman for permission to examine cases at the Hospital for Sick Children, Great Ormond Street, and to Mr. Stansfield Collier for permission to make use of one case.

This investigation is concerned with :—

(1) The nature and variations of the plantar reflex in normal persons.

(2) The character of the plantar reflex in diseases of the

nervous system, and the clinical value of deviations from the normal in such conditions.

(3) A theory of the causation of pes cavus in spastic states: it being possibly a result of reflex hypertonicity preponderating in certain muscles.

Method of Clinical Investigation.

The supine position with the lower limb semi-flexed at the hip and knee and rotated out with the knee resting on a pillow so as to ensure muscular relaxation, was found to be the best for the observation of the reflex. The position of the limb was not found to have any effect upon the form of the reflex. Every precaution was used to get rid of expectant attention on the part of the person examined by distracting his attention by conversation or by the performance of some act with his hands requiring attention.

All cases in which, on repeated examination, it was found impossible to exclude volitional movements, or whose movements were not constant, or where the movements were too extensive to admit of careful investigation, were excluded from this investigation. Such cases were only rarely met with. Except in the normal cases, repeated observations were made upon each subject to ensure that the signs were constant.

Observations were also made during sleep upon many of the cases.

The plantar region was stimulated by gently scratching with the finger nail or with a soft quill, minimal stimuli being always used where possible. The effect of increasing the stimulus gradually to a heavy scratch with the nail, and the effect of painful stimulation was observed.

The order of response of the muscles in the reflex was recorded (1) by means of levers resting at one end upon that part of the muscle which bellied most during contraction, and at the other end resting upon a travelling surface; (2) by strings attached to the toes and each pulling upon one arm of a weighted bell crank lever.

The afferent path in this reflex is by the fifth lumbar

and first sacral roots. I have observed that in persons where the plantar reflex was very easily elicited, stimulation of the skin over the dorsum of the foot and over the outer side of the leg (also supplied by the fifth lumbar root) produced movements precisely similar to those elicited by stimulation of the sole of the foot. The same result was never obtained from stimulating the third and fourth lumbar or second and third sacral root areas.

The response is usually confined to the lower limbs, but the involvement of the abdominal and back muscles in the response is frequently seen in children, and is much more marked on the side of stimulation.

The ease with which the plantar reflex is elicited and the degree of response varies greatly in different individuals, but the nature of the reflex is remarkably constant. I have not found it constantly absent in a healthy person. Coldness of the feet will temporarily abolish it or make it very difficult to obtain. Sensible perspiration on the feet has the same effect. Moisture does away with much of the element of friction so necessary for the production of the skin reflexes. Its effect may be readily demonstrated by moistening the back of one's hand: it becomes much less "ticklish." I have in many instances, by carefully drying the moist feet and warming the cold ones, obtained a good plantar reflex where I had failed previously. Light scratching or tickling stimuli are much more efficacious in producing the plantar reflex than more severe and painful stimuli, and the former will often succeed where the latter fail. I have not found the nature of the reflex to change in any way except in degree with different kinds of stimulation.

The irritability of the different skin regions in the sole of the foot varies greatly in individuals. I have not been able to make out definite areas where the plantar reflex could be obtained more readily, such as have been classified by Brissaud.[3]

In the clinical investigation of the plantar reflex it is very important that all the muscles of the lower limb should be observed at the moment of stimulation, because, as will

be subsequently stated, the earliest response is generally in muscles of the hip, and in cases where the plantar reflex is difficult to obtain, the only response may be in those muscles.

This last phenomenon is frequently met with in functional disease; in peripheral neuritis and in tabes dorsalis.

In certain cases the reflex only appears upon the first one or two stimulations and is then absent even on the strongest excitation. This is especially apt to occur in functional cases with a slight reflex.

If the preceding points be taken into consideration, I think that the common statement that the plantar reflex is not rarely found completely absent in healthy subjects[4] will not be confirmed.

The Normal Plantar Reflex in Adults.

If a minimal stimulus be applied to the sole of the foot, a contraction of the tensor vaginæ femoris,[3] often accompanied by a less marked contraction of the adductors of the thigh and sartorius is observed in the great majority of cases.[5] With a slightly stronger stimulus flexion of the four outer toes, the movement preponderating towards the little toe appears. With increasing stimuli all the toes are flexed on the metatarsus and drawn together, the ankle is dorsiflexed and inverted; the knee is flexed and the hip is strongly flexed, adducted and rotated inwards so as to carry the knee across the opposite thigh. With still stronger stimulation violent irregular movements of the limbs occur which spread to the lower part of the trunk and to the opposite limbs.* The position of the foot at the height of a response to a moderate stimulus is shown in fig. 1. In some cases, which, however, constitute an inconsiderable minority, flexion of the four outer toes occurs with the same or even a smaller strength of stimulus than does contraction of the tensor fasciæ femoris. There is then in

* In a case of anterior poliomyelitis with complete palsy of the lower limbs, the most gentle stimulation of the sole of the foot produced a vigorous single twitch in the levator ani.

response to increasing stimulation of the sole of the foot, a definite order of succession in the contraction of the muscles which respond, and the contraction is more vigorous in those muscles which respond earlier than in those which react later. To illustrate this last fact the behaviour of the calf muscles may be stated. They may be felt to contract only to considerable stimulus, yet although they are much stronger muscles than the anterior tibial group, they never overcome the dorsiflexor movement, and plantar extension of the ankle is never seen in the plantar reflex of a normal person.

The order of contraction in all the normal cases was found as follows:—

(1) Contraction of the tensor fasciæ femoris, sartorius and adductors, and flexion of the hip.

(2) Flexion of the four outer toes.

(Sometimes 1 and 2 are simultaneous, and rarely the order is reversed.)

(3) Dorsiflexion of the ankle.

(4) Flexion and adduction of all the toes; inversion of the foot by the tibialis posticus; flexion of the knee.

(5) Contraction of calf-muscles.

(6) Contraction of quadriceps.

The early response of the tensor fasciæ femoris in the plantar reflex was first pointed out by Brissaud,[3] and it was called by him "le reflex du fascia lata."

As the response in the tensor fasciæ femoris is the first to appear, so also is it the last to disappear when the plantar reflex is diminished in normal subjects by cold, anæsthesia, and unknown factors, which cause this reflex to vary in degree from time to time in healthy subjects. I have several times found a lively response in this muscle when no other muscle reacted to stimulation of the plantar region.

The terms "early" and "late" response are here used with reference solely to the time at which a muscle reacts to stimuli, gradually increased from the minimal stimulus capable of producing a plantar reflex, and not with any reference to actual "reflex time." I have, by means of tracings taken directly from the muscles, observed that the

reflex times of the several muscles in the plantar reflex only vary to an extent that can be accounted for by the different lengths of the nerves supplying them, although from the different amounts of contraction, certain muscles seem to follow others on superficial observation.

The movement produced by a moderate stimulus preponderates, as has been above explained, in the hip and ankle, and on account of the greater weight that the hip-muscles have to raise in contracting, it is most conspicuous at the ankle. This is a striking point of difference between the normal plantar reflex and that to be subsequently described in certain lesions of the central nervous system.

The economic significance of the nature of this reflex is clear. In plantigrade progression, if an irritating object come in contact with the sole of the foot, the flexors of the hip acting at greater mechanical advantage as regards range of movement than any other muscle, will be best adapted to remove the foot from the offending object; the foot is fixed by the dorsiflexion of the ankle, and the whole surface is at once removed from the ground, while the flexion of the toes and the inversion of the foot render the skin of the plantar region lax, so that any sharp object will penetrate less readily.

For the sake of brevity in this communication I propose to call this normal form of plantar reflex characterised by flexion of the toes and marked early action of the tensor fasciæ femoris as the "flexor response."

The Crossed Plantar Reflex.

A crossed plantar reflex can usually be obtained in cases where the plantar reflex is marked. It consists of a slight movement of flexion of the toes, especially of the great toe, with slight dorsiflexion of the ankle.

Usually a much stronger stimulus is necessary to produce the crossed plantar reflex than the direct, but this is not always so; sometimes the minimal stimulus required to produce the plantar reflex will evoke a distinct crossed reflex.

The Normal Plantar Reflex of Infants.

The response is very brisk, and differs very markedly from the adult reflex. The earliest response is in the great toe, which is drawn back. This is followed by extension and spreading out of all the toes with eversion of the foot and dorsiflexion of the ankle, and subsequently flexion of the hip and knee. Strong stimulation causes a general, irregular movement of the limbs and trunk. The crossed plantar reflex, when I have been able definitely to obtain one, has consisted of extension of the toes.

This form of reflex, with the widely-spread, drawn-back toes and the late response of hip-muscles contrasts most markedly with the adult form, with the contracted, flexed toes, inverted foot and the early hip response. I propose to use the term "infantile response" for this type of plantar reflex.

It may be remarked here that this form of plantar reflex is precisely similar to the plantar reflex of such monkeys as I have examined, and may be considered a more primitive type. To this point I shall recur subsequently.

The infantile response persists until the child becomes able to walk, when it is gradually replaced by the flexor response. I have observed several cases in the transition period, and in these children sometimes one and sometimes the other form of response occurred at the same examination, or a mixed response of flexion of the outer toes with marked extension of the great toe occurred.

The age at which the change occurs is generally between the second and third year. I have met with the typical adult response in children of two years old, who were particularly forward and "strong on their legs."

In weakly, and especially in rickety children, the infantile response may be seen as late as the age of four years.

It may, then, be stated that with the establishment of complete cerebral control over the lower limbs the adult form of response to the plantar reflex replaces the infantile response.

The Plantar Reflex during Sleep.

During sleep the plantar reflex is diminished, and in infants and adults the infantile and flexor responses occur respectively, but in some children up to the age of twelve years who presented the adult flexor response when awake I have observed a most interesting phenomenon. During deep sleep there was a typical infantile response, with the extension and spreading of the toes and eversion of the foot. With repeated stimulation complicated co-ordinated movements directed towards the removal of the cause of irritation were noticed. It seemed as if some higher mechanism had been roused into action. Further irritation of the foot caused the child to awake, and directly he did so the normal adult plantar reflex occurred on stimulation.

In these cases the temporary loss of cerebral influence over the spinal cord during sleep was accompanied by a reversion to the primitive infantile response, which disappeared and was replaced immediately by the adult response on awakening.

In children between the ages of six and twelve years this peculiarity was only found in a minority of the subjects examined.

The Plantar Reflex in Pathological Conditions.

(1) Lesions of the pyramidal system.

An alteration in the form of the plantar reflex in these lesions was first described by Babinski.[1] He found that if in the plantar reflex, extension of the toes, especially of the great toe, upon the metatarsus occurred, this extension of the toes was pathognomonic of a lesion of the pyramidal system. To this rule he made two exceptions: (1) In newly-born infants he noticed that the infantile response was constant; (2) In cases of total transverse lesions of the cord he was unable to obtain any plantar reflex.

He named this sign "le phénomène des orteils," and

states that he has never found it in healthy subjects or in functional cases, but only where there was clinical evidence of a lesion of the pyramidal system, and conversely that in lesions of the pyramidal system, with the exception of total transverse lesions of the spinal cord, this sign almost invariably occurs. His pathological investigations entirely confirmed this statement.

Van Gehuchten [2] has confirmed these results.

I find that the form of plantar reflex in lesions of the pyramidal system, whilst resembling the infantile response in many respects, differs in a few points. In a well-marked case, the whole response is more deliberate than in the flexor response or the infantile response, and appears with much more certainty than does the flexor response to each stimulation. Extension of the great toe precedes all other movement. It is followed by extension of the other toes and by dorsiflexion and inversion of the ankle. The tensor fasciæ femoris does not contract early. The tibialis posticus and anticus and peroneus longus contract strongly, and fix the ankle, and a temporary increase of the plantar arch is often noticed. The small amount of movement at the ankle in the reflex from this cause is conspicuous, and contrasts with the brisk movement of the ankle in the normal reflex. There is often a tendency of certain muscles to remain contracted after the reflex, especially the extensor proprius hallucis, reminding one of the cataleptoid reflexes observed by Sherrington in paraplegic monkeys.[6]

Just as the response of extension of the great toe is the first to appear, so when the reflex is very sluggish the extensor proprius hallucis may be the only muscle to respond even to strong stimulation.

I propose to use the term "extensor" response for this form of reflex.

This extensor response is often more easily elicited by stimulation of the outer part of the sole, whereas the flexor response is usually more easily obtained by stimulating the inner part.

The position of the foot at the height of a moderate extension response is depicted in fig. 2.

FIG. 1.

The position of the foot of a healthy adult at the height of a flexor response. The flexion and adduction of the toes, and the dorsiflexion and inversion of the ankle are well shown. (From an instantaneous photograph.)

FIG. 2.

The position of the foot at the height of a moderate extensor response. The increase of the plantar arch and the extension of the toes, especially of the hallux, are well shown. From an instantaneous photograph. The case was one of early disseminated sclerosis.

In the following table the chief characteristics of the three forms of response are contrasted.

	Normal.	Extensor Response.	Infantile Response.
Movement	Quick	Often deliberate	Quick
Muscle first to contract with a minimal stimulus	Tensor fasciæ femoris	Ext. prop. hall.	Ext. of toes
Certainty of response to each stimulus	Less certain	Certain	Less certain
Position of toes	Flexion; adduction	Extension; spreading	Extension; spreading
Obtained more easily by stimulating	Inner part of the sole	Outer part of the sole	
Movement of ankle	Dorsiflexion and inversion, both conspicuous	Dorsiflexion and inversion, both slight	Dorsiflexion and eversion, both conspicuous

According as the lesion of the pyramidal system be partial, the flexor and extensor responses may be combined in the plantar reflex. The following are common clinical variations:—

(1) Extension of great toe with flexion of four outer toes.

(2) Slight flexion of all toes, followed by slow, strong extension.

(3) Contraction of tensor fasciæ femoris, and of extensor proprius hallucis with the minimal stimulus.

(4) Stimulation of the sole may sometimes produce the flexor, and sometimes the extensor response.

Especial care must be taken in investigating the plantar reflex that a movement of dorsiflexion of the ankle is not mistaken for a movement of extension of the great toe. The movement at the metatarso-phalangeal joint must be carefully inspected to obviate this source of error.

The Crossed Plantar Reflex Associated with the Extensor Response.

The cases showing the extensor response almost invariably present slight flexion of the toes, preponderating in the great toe as the crossed plantar reflex, whether the condition present is a hemiplegia or a paraplegia.

I have met with slight extension of the toes—chiefly of the outer toes—as a crossed plantar reflex in two cases only, both cases of severe spastic paraplegia.

The clinical and pathological results obtained in this investigation have, for the most part, confirmed Babinski's conclusions, and exceptions to his rule that the extensor response indicates a lesion of the pyramidal system have been found so few as to "prove the rule."

(1) *Total Transverse Lesion of the Cord.*

In three cases of total transverse lesion of the cord I have found the extensor response present. Bastian[7] states that in his cases slight movements of the toes on stimulating the sole were the only reflex phenomena present, but does not state the nature of the movement.

Case I.—R. L., labourer, aged 52 years, was admitted into St. Mary's Hospital, under Mr. Stansfield Collier, on September 26, 1898, with a severe injury to the dorsal spine, caused through a fall a few hours previously. He was completely paraplegic, and the knee-jerks were absent. A laminectomy was at once performed, and the spinal cord was found completely severed at the level of the fifth dorsal vertebra. Twenty hours later stimulation of the sole of either foot produced a sluggish extension of all the toes, especially the great toe, and this sign persisted till his death three days later. The response was absolutely confined to the foot. The knee-jerks were persistently absent.

Case II.—A. G., aged 50 years, was admitted into the National Hospital under Dr. Buzzard suffering with cancer of the dorsal vertebræ secondary to a scirrhus of the left breast, which had been removed. She had complete flaccid paraplegia and complete anæsthesia as high as the eleventh dorsal root

level. The knee-jerks were absent and the sphincters completely incontinent. Up to the time of writing, these physical signs have been constant, except that the anæsthesia is creeping upwards. She has always shown a moderate extensor response at the plantar reflex.

(Since writing the above the patient, A. G., died. At the autopsy a mass of new growth was found compressing the spinal cord at the level of the seventh dorsal nerve-root. The cord was narrowed to the size of a crow-quill and its substance was semi-diffluent at this point.)

Case III.—A. E., a laundress, aged 20 years, was admitted into the National Hospital under Sir William Gowers on Sept. 16, 1898, suffering with acute syphilitic transverse myelitis of six days' duration. She had complete flaccid paraplegia and absolute anæsthesia up to the tenth dorsal root level; complete incontinence and loss of knee-jerks. The plantar reflex was a marked slow extension of the hallux alone. The knee-jerks returned ten days later, and some movement fifteen days later.

Although there was no anatomical proof of a total transverse lesion in the last two cases, there was full clinical evidence of a physiological total transverse lesion.

(2) *Myelitis* (*including Compression Paraplegia*).

Of thirty-six cases repeatedly examined, thirty-four always showed a marked extensor response. In one case of twenty years' duration there was great organic contracture of the calf muscles, so that the ankles were fixed in a position of plantar extension and could not be passively dorsiflexed; the toes were fixed in a position of flexion and could not be extended. The faradic excitability of the anterior tibial muscles was absent. The plantar reflex was confined to a flexion of all the toes.

In one case of cervical myelitis there was associated atrophic palsy of the extensors of the toes and loss of the deep reflexes. The plantar reflex consisted in a flexion of the toes.

The two following cases exemplify the return of the plantar reflex to the normal on recovery.

Case IV.—Percy C., aged 5 years, was admitted into the National Hospital under Dr. Tooth on April 15, 1898, with symptoms of spinal caries of six weeks' duration and gradually increasing paraplegia of three weeks' duration, with hesitating micturition, increased knee-jerks and double foot clonus. The plantar reflexes were very strong extensor responses.

On May 17, having been treated with rest and extension since admission, he was completely paraplegic. On May 17 Mr. Horsley performed laminectomy and removed a large amount of gelatinous tubercular material that was found pressing upon the theca.

On June 7 he began to move his legs. The wound was healed on June 25.

On October 20 he could walk well, and with the exception of some increase of the knee-jerk, all his symptoms of paraplegia had disappeared. The plantar reflex was sometimes an extensor, and sometimes a flexor response.

On October 30 the plantar reflexes were constantly flexor responses. He could walk and run naturally.

Case V.—J. P., aged 34 years, an engineer, was admitted into the National Hospital under Dr. Beevor in November, 1898, with spinal caries and slight weakness of the legs. Movements of the legs were well performed, but with a feeling of stiffness. The knee-jerks were increased, and there was a little foot clonus. He had occasional reflex flexor spasms in the legs. The plantar reflexes were marked extensor responses; the extensor proprius hallucis contracting to a much slighter stimulus than did the other muscles concerned in the response.

After a fortnight's rest in an extension apparatus, the feeling of stiffness, increased deep reflexes and flexor spasm had entirely disappeared. The plantar reflex, after showing sometimes an extensor response and sometimes a flexor response, became constantly a flexor response.

In these cases the extensor response was the last of the "spastic" signs to disappear.

(3) *Disseminated Sclerosis.*

Of thirty cases, twenty-eight showed an extensor response on both sides. In one case in which the signs were limited to the right limbs, there was an extensor response on the right side and a flexor response on the left.

In one case in which the knee-jerks were diminished and the chief symptom ataxy, there was a flexor response on both sides.

In five cases of paraplegia, where the symptoms had been entirely those of functional disease and a diagnosis of functional paraplegia was made, symptoms of organic disease, such as diplopia, sphincter trouble, erythromelalgia, girdle sensation, &c., subsequently appeared, and the diagnosis was changed to (?) disseminated sclerosis.

In two of the cases the onset of unequivocal signs of organic disease was accompanied by a change from the flexor to the extensor response and persistence of the latter.

In two cases the flexor response persisted, though signs of extensive organic disease developed.

In one case the tensor fasciæ femoris alone responded in the plantar reflex.

In the differential diagnosis between functional and organic hemiplegias and paraplegias in adults, the presence of an extensor response is a most valuable sign of organic disease, such as is not furnished by increase of the knee-jerks or by the less persistent forms of foot-clonus. The presence of a flexor response, however, is compatible with the presence of organic disease.

(4) *Hemiplegia.*

Of thirty-six cases of organic hemiplegia, twenty-eight gave an extensor response on the paralysed side and a flexor response on the sound side. The reflexes were found in some cases very brisk, in others very difficult to obtain. The cases with brisk and those with sluggish reflexes were classified and compared, but no general fact as to the cause of the difference was elicited.

In three cases the extensor response was found on both sides, and an autopsy in one of them—a case of right hemiplegia—revealed degenerative changes in both hemispheres.

In one case of eight weeks' duration the plantar reflex

was entirely absent on the paralysed side, a flexor response being present on the sound side.

In four cases the flexor response was found on both sides. In one of them the leg had entirely escaped the paralysis. In the other three the flexor response was more marked on the paralysed side and was very atypical, consisting of a vigorous flexion of all the toes. In these three cases the appearance of the foot on the hemiplegic side was peculiar; the foot was dropped, and the toes were, when at rest, bent strongly towards the sole of the foot, the line of the inner side of the foot from the tip of the great toe to the heel forming the chord of a circle. This position of the foot is the opposite to that of pes cavus, which is not rarely met with in cases where the pyramidal system is damaged, and which will be subsequently described as having a most important connection with the extensor response.

I have no explanation to offer for the peculiarities of the plantar reflex in these three exceptional cases.

(5) *Friedreich's Disease.*

Of six cases all showed a marked extensor response. Four of the cases had marked pes cavus, and the knee-jerks were absent. The others had slight pes cavus, and the knee-jerks were just obtainable.

Clinically, the only sign of the affection of the lateral columns is the extensor response in the plantar reflex and the pes cavus.

(6) *Syringomyelia.*

Of three typical cases all showed a marked extensor response.

(7) *Amyotrophic Lateral Sclerosis.*

Of three cases all showed an extensor response.

(8) *Bulbar Palsy and Progressive Muscular Atrophy.*

The cases in which the deep reflexes in the lower limbs were increased showed the extensor response. Where the lower limbs were normal the flexor response was present.

Epilepsy.

Of forty cases thirty-eight showed the flexor response. After every fit of severe convulsions lasting any length of time the extensor response was found present, and lasting some time.

In one patient, who suffered from periodic groups of attacks of severe general convulsions, the flexor response was constantly present when he was free from attacks. During a period of forty hours he had fourteen severe fits; the extensor response was always present and persisted for seven hours after the last attack, when the flexor response returned.

The extensor response, as a rule, was present for from five to ten minutes after a general convulsion.

In cases of hemi-epilepsy, not associated with hemiplegia, the extensor response was only present after the fits on the convulsed side.

Where the general convulsions were of very brief duration often no change in the plantar reflex was observed.

Cases of Jacksonian epilepsy affecting the foot always presented an extensor response for a short time after the fit.

The following two cases of epilepsy were exceptional:—

Case VI.—A. S., aged 20 years, under the care of Dr. Bastian, had suffered with right hemi-epilepsy since a severe fit at the age of two years. The right arm and leg were distinctly smaller than the left, and the movements, although all well performed, were slightly weaker than those on the left side. The gait was natural, and the right knee-jerk was only slightly more lively than the left. She had had no fit for six weeks before admission. The right plantar reflex was constantly an extensor response, the left a flexor response.

In this case a lesion of the left cortex had probably occurred at the time of the first fit. The extensor response was the most evident sign of the hemiplegia, the weakness and impaired development being so slight as to be easily overlooked.

Case VII.—J. G., aged 28 years, under the care of Dr. Ferrier, had suffered for two years with slight general convulsions.

She had not had infantile convulsions. There was no physical sign of organic disease, except that an extensor response was constantly obtained in the left foot, a flexor response in the right.

I look upon this case with suspicion, and think that it may possibly be a case of cerebral tumour.

Many patients have presented in the out-patient department of this hospital as cases of idiopathic epilepsy, who for years showed no sign or symptom of organic disease, and subsequently developed unequivocal signs of intracranial tumour. Several such cases subsequently examined in the *post-mortem* room have shown neoplasms in the anterior parts of the hemispheres.

I would suggest that the presence of a constant extensor response, since it may be the earliest sign of a damage to the pyramidal system, may be of great value in the early recognition of some of these cases.

In the following case of cerebral tumour, commencing with epilepsy, the presence of a unilateral extensor response was of great clinical value.

Case VIII.—Annie S., aged 12 years, was admitted on Sept. 22, 1898, into the National Hospital, under the care of Dr. Buzzard.

Four months before admission, after an attack of influenza, she began to have frequent attacks of "petit mal." She would suddenly turn pale and look strange, and say "I feel so funny." A fortnight later it was noticed that in some of the attacks the left arm twitched.

She was sent to the Meath Home for Epileptics, and when admitted there she was constantly falling asleep at all times of the day, though quite bright and natural when awake. It was then noticed that she did not use the left side of her face in talking so well as the right.

A month before admission her sight began to fail rapidly. A fortnight later paroxysms of vomiting began for the first time in her illness. She had no headache until two days before admission. On admission she was obviously very ill. Her mental state was intelligent. Speech and articulation were normal. She had frequent Jacksonian attacks, beginning sometimes in the left face, sometimes in the right face, sometimes in

the right hand and sometimes in the left hand, remaining always confined to the side on which the attack commenced. She was quite conscious and able to speak during the attacks. Taste, smell and hearing were normal. There was intense optic neuritis showing a commencing subsidence into atrophy. There was absolutely no light perception. There was complete palsy of the right third and sixth nerves and partial palsy of the left third and sixth nerves. There was very slight weakness of the left lower face and of the left arm. There was absolutely no weakness of the legs, and her gait was natural. There was no localised cranial pain or tenderness. Sensation was natural. The knee, wrist and elbow jerks were absent and were never obtained up till her death. The plantar reflex on the right side was a marked flexor response, that on the left side a vigorous extensor response. This condition of the plantar reflex persisted till the day before death, two months later, when she became deeply comatose, and the extensor response was present on both sides, but was much more marked on the left side.

A month after her admission she dragged the left foot slightly in walking, but after this the hemiplegia did not progress.

The autopsy revealed an infiltrating tumour of the white substance of the right temporo-spenoidal lobe extending as far as the right capsule. A tongue of new growth extended over the right crus and was pressing upon the right third nerve.

In this case the early attacks of "petit mal," the somnolence and the Jacksonian attacks indicated a lesion of the hemispheres, but did not suggest which hemisphere was involved.

The slight hemiplegic symptoms in the left face and arm confirmed by the extensor response in the left foot gave an accurate indication as to the side of the lesion.

Cerebral Tumour.

Sixteen cases were examined in which there were no physical signs of any lesion of the pyramidal system. In all of these the flexor response was present.

Cerebellar Tumour.

Of three cases all showed a flexor response.

Functional Disease.

Of thirty-five cases, fifteen showed a moderate flexor response. In three cases there was an extremely brisk flexor response on the least stimulation of the sole. In ten cases the flexor response was elicited with difficulty.

In seven cases the plantar reflex had been recorded in the notes as absent, and in two of them this absence had been repeatedly noticed for years, but in all of these cases there was a distinct contraction of the tensor fasciæ femoris on stimulating the sole. The reflex only followed the first few stimulations, and then disappeared. It was apparently very easily tired out, for it always reappeared after a few minutes' rest.

The absence of the plantar reflex in cases of functional paraplegia where there is no anæsthesia of the feet has been brought forward by Dr. Buzzard[8] as characteristic of that condition. So far as the absence of response in the foot is concerned, it occurred in 20 per cent. of all cases examined and almost invariably in functional paraplegia, but I did not meet with a functional case in which there was entire and constant absence of response in the muscles of the hip.

Paralysis Agitans.

Of ten cases, the flexor response occurred in all, and the reflex was sluggish.

Tabes Dorsalis.

Of twenty-five cases, eight showed an entire and persistent absence of any response. In three cases the tensor fasciæ femoris alone responded. In five cases an attempt to elicit the plantar reflex produced irregular movements of the toes and foot, often of considerable range lasting some time. In the remaining cases the flexor response was present, in some cases much delayed, in some cases brisk, in others sluggish. Anæsthesia of the feet seemed to have no connection with the presence or absence of the reflex. In one case where there was long-standing complete anæsthesia to all forms below the knees the flexor response was particularly brisk.

The extensor response was never met with.

In the following case the presence of a marked extensor response furnished a valuable suggestion as to the diagnosis.

Mary C., aged 35 years, married, was admitted into the National Hospital under Dr. Ferrier on September 28, 1898, complaining of weakness of the legs and inability to walk. Her previous health had been good. Her first child was born dead at six months, and her only other child was alive and healthy. Her illness commenced eighteen months before admission with subjective stiffness in the calves, tingling of the feet and a tight girdle sensation. Nine months before admission she dragged both feet in walking, and had slight precipitancy of micturition. Six weeks before admission she became entirely unable to walk alone.

The pupils and cranial nerves, optic discs and special senses were normal. There was slight blunting of sensibility over the feet as high as the ankles, some loss of sense of position at ankles and knees. She complained of aching pains in the legs. There was no wasting of the lower extremities and all movements were performed with moderate power. There was no hypotonia. There was great ataxy in the legs and marked Romberg's sign. In walking the feet were poorly lifted, but did not scrape the ground. The knee-jerks were absent. There was some precipitancy of micturition. The plantar reflex on both sides was a marked extensor response.

The case presented at first sight as one of atypical tabes but there was a definite history that she had dragged her feet in walking before she became very ataxic. This was not a symptom of posterior sclerosis.

In the cases of tabes examined by Babinski, Van Gehuchten and myself, the extensor response was never met with.

Babinski states that in his cases presenting the extensor response every autopsy revealed a lesion of the pyramidal system, and the same result had been obtained from autopsies upon my cases.

From these facts the diagnosis of postero-lateral sclerosis was in this case strongly suggested.

(Since writing the above two patients who presented as atypical cases of tabes, the knee-jerks being lost and the extensor response present, as in this case, have died in the

National Hospital. Autopsies upon both cases revealed marked degeneration in both the posterior and lateral columns of the spinal cord.)

Multiple Peripheral Neuritis.

Of fifteen cases nine gave a flexor response, generally sluggish. In three severe cases the plantar reflex was entirely absent while the patients were under observation. Three cases showed a response confined to the tensor fasciæ femoris.

Chorea.

In twenty cases (including three cases of Huntingdon's chorea) a normal flexor response was found.

Anterior Poliomyelitis and Myopathies.

In twenty cases of acute poliomyelitis, occurring after the patients had learnt to walk, the flexor response was met with. Where the muscles concerned in the response were involved in the palsy, the response was diminished or lost in proportion to the degree of paralysis.

In six cases of myopathy a similar result was obtained. One case, however, was exceptional. This was a very typical case of pseudo-hypertrophic palsy in a boy of nine years old, who had learnt to walk when eighteen months old, and had been particularly strong and active upon his legs till the age of four years, when his symptoms commenced. In this boy a marked infantile response was always present.

In cases of *neurasthenia* and *sciatica* the plantar reflexes were found normal.

THE INFLUENCE OF STRYCHNINE AND TETANUS TOXINE ON THE PLANTAR REFLEX.

M. Babinski has recorded a case of strychnine poisoning in which "le phénomène des orteils" was present and disappeared soon after recovery.

Several adults who presented the flexor response have been treated under my care with increasing doses of a

solution of nitrate of strychnine administered hypodermically and I have found that when large doses—one-sixth to one-third grain—are given, the form of the plantar reflex becomes changed, and resembles that found in lesions of the pyramidal system.

The change in the reflex was noticeable about five minutes after the injection, and persisted for about half-an-hour. No discomfort beyond a slight feeling of general stiffness on movement was experienced by the patients after the injection.

The same change persisting many weeks after recovery was observed in a case of tetanus recently in the National Hospital.

Case X.—S. B., a healthy boy, aged 5 years, was admitted under the care of Dr. Hughlings Jackson, on September 6, 1898. Tetanic symptoms had set in ten days after a wound on the forehead. The spasms were severe and confined to the head, neck, and upper limbs. They ceased on the tenth day after admission, and he became rapidly convalescent. During the stage of acute symptoms, and for a month afterwards, a strong, deliberate extensor response was invariably the result of stimulating the sole of the foot. On October 24 sometimes a flexor response, and sometimes an extensor response, with a diminished contraction of the tensor fasciæ femoris were obtained. After November 1 no extensor response was obtained, a marked flexor response being present, with a very lively action of the tensor fasciæ femoris. This form of reflex persisted as long as he was under observation.

Both strychnine and tetanus poison increase the reflex excitability of the spinal cord, and the cerebral control is thereby relatively lessened. The increased reflex excitability was in these cases accompanied by a return of the form of plantar reflex to the infantile type.

The Ætiology of Acquired Pes Cavus in Spastic Conditions.

In cases of Friedreich's disease, in the family type of primary lateral sclerosis, in lesions of the spinal cord producing a spastic condition of the lower extremities, and in

cases of hemiplegia (especially in children and young people) a deformity of the foot known as pes cavus is sometimes met with. In well-marked pes cavus the whole foot is dropped; the toes, especially the great toe, are retracted, forming an angle with the metatarsus. The metatarsus itself is pulled down in front, and with the anterior part of the tarsus forms an angle with the posterior part of the tarsus, so that the inner border of the foot has an outline somewhat resembling that of the letter *Z*.[11] The plantar arch is increased and the foot is inverted. In long-standing cases the metarso-tarsus is often structurally shortened.

Several explanations have been brought forward to account for the development of pes cavus. Duchenne[9] considered that it is due to weakness or paralysis of the interrossei, combined with weakness of the dorsiflexors of the ankle, and stated that weakness of these muscles and an altered electrical excitability may be found in them. He considered pes cavus as a similar condition with a like cause to "main en griffe."

Golding Bird[10] also considers this explanation valid.

If a hand presenting "main en griffe," with its paralysed and atrophied muscles, structural rigidity, and marked rigid flexion of the interphalangeal joints be compared with a foot showing marked pes cavus in Friedreich's disease, where there is no wasting nor paralysis, no contraction of tendons, no rigid flexion of the interphalangeal joints, and no alteration of electrical excitability in the muscles, the resemblance between the two will be found superficial rather than real.

I have carefully examined many cases of pes cavus, and have been unable to find any relative weakness of these muscles, or any change in their electrical excitability.

Duchenne's explanation applied to the pes cavus of spastic states is, I think, untenable, and does not account for the inversion of the foot, nor adequately for the increase of the plantar arch.

Adams,[12] in 1864, considered the condition to be produced by spasm in certain muscles.

Barwell,[13] in a recent paper, considers Duchenne's ex-

planation will not account for most of the cases. After discussing the congenital and paralytic forms, he says, "a third class of pes cavus is due to spastic tension in the long peroneal muscle. Though I cannot determine the cause of this contraction, I have more than once demonstrated its presence."

I have examined a large number of cases of hemiplegia and paraplegia of long duration, with spastic lower extremities, and have found in all of them which showed an extensor response in the plantar reflex a tendency to that position of the foot known as pes cavus. The results were most striking in cases of hemiplegia, where the non-affected foot could be taken as the normal. (Accurate measurements of the length of the plantar arch, &c., were taken by means of a flat rule placed against the sole of the foot and a divider working at right angles to the rule. The measurements were also taken from accurate plaster casts of the same feet in some cases.)

I have been able to watch the development of pes cavus from a normal foot in three cases. The feet depicted in figs. 3 and 4 were those from a case of spinal caries with paraplegia, under the care of Dr. Bastian. This patient was admitted into the National Hospital with a condition of slight spastic paraplegia, and her feet showed no deformity; she became absolutely paraplegic, and pes cavus slowly developed. The photograph was taken a year after the onset of the paraplegia.

I have also had the opportunity of examining four cases of early Friedreich's disease with commencing pes cavus. In all these cases the earliest signs of deformity was retraction of the great toe, and the second noticeable event was spasm of the tibialis posticus and peroneus longus muscles, as evidenced by the constant standing out of their tendons behind the internal and external malleoli respectively.

In a well-marked case, the standing out of the tendons of the extensor proprius hallucis, tibialis posticus, and peroneus longus is most noticeable, and is well depicted in figs. 4 and 5 (in this case there was no structural rigidity; the deformity could be easily reduced by force). Exception

must here be made in cases of late Friedreich's disease where considerable hypotonia has developed.

A condition of muscular spasm preponderating in the extensors of the toes, tibialis posticus and peroneus longus would produce a position of the foot similar to pes cavus. The toes would be extended, the forward part of the metatarsus would be drawn down by the tendon of the peroneus longus attached to the base of the first metatarsal bone, and by the prolongation of the tibialis posticus to the middle three metatarsal bones. Both of these muscles increase the plantar arch, and both plant-extend the foot. Inversion of the foot would result, as the tibialis posticus is a stronger muscle than the peroneus longus, and inversion of the foot a stronger movement than eversion.

Isolated paralysis of the tibialis posticus produces entire loss of the antero-posterior plantar arch and eversion of the foot. (Erichsen.)[14]

I would suggest that spasm preponderating in the muscles is the correct anatomical explanation of acquired pes cavus, and there is clinical evidence that such spasm does actually exist in these cases.

A physiological explanation for the occurrence of such spasm must be sought for, and it will be found in the extensor response in the plantar reflex. Pes cavus occurs not infrequently in diseases in which a lesion of the pyramidal system occurs, and I think that a tendency to pes cavus may be found in nearly all such cases if they be of long standing. The extensor response is almost always present in such cases, and is especially marked when any degree of pes cavus occurs.

The tone of the muscles supplied by a spinal segment is dependent upon the reflex arc of that segment, and may be described as the response on the efferent side to the continuous afferent impressions reaching the segment by its afferent nerves.

In the absence of the control of higher centres the physiological capacity for reflex response of the spinal segment increases, and with it the muscular tone which may go on to rigidity. The efferent impulses leaving the

FIG. 3.

The appearance of the left foot at rest from the internal aspect in a case of spinal caries under the care of Dr. Bastian. The pes cavus had developed while the patient was in hospital. The standing out of the tibialis posticus tendon is seen. (From a photograph.)

FIG. 4.

The right foot from the same case as Fig. 3, seen from the outer side, showing the standing out of the tendon of the peroneus longus.

spinal segment, in the absence of control from higher centres, will be distributed to muscles in the same pattern as the response to the superficial reflex connected with that segment.

The form of the superficial reflex in the foot, when the higher centres are cut off, is, extension of the toes, with slight inversion and dorsiflexion of the foot, and increase of the plantar arch, and marked action of the tibialis posticus and anticus and peroneus longus. The pattern of muscular tone in the same condition is this extensor response stereotyped; there is relative hypertonicity in the extensors of the toes, in the tibialis posticus and peroneus longus, and to a less degree in the tibialis anticus. This relative hypertonicity, if persisting for a long time, will cause the foot to assume the position of more or less marked pes cavus. The deformity will be more marked in young people since the forces producing it act upon less rigid skeletal structures and during a period of growth, than in old people where the structures are more rigid.

(The physiological basis of the muscle tone and of rigidity is probably far more complicated than stated above, but the explanation given, so far as it goes, quite suffices for the case at issue.)

As a strong argument in favour of the truth of this explanation of pes cavus the three exceptional cases of hemiplegia with a flexor response upon the affected side may be again mentioned. The deformity of the affected foot in these cases resembled the position a normal foot assumes at the height of a vigorous flexor response, just as pes cavus resembles the position of a foot at the height of an extensor response.

Conclusions.

(1) This investigation has confirmed those of Babinski, Van Gehuchten and Brissaud in all essential points.

(2) The plantar reflex of healthy subjects, with the exception of infants, consists of a regular succession of contraction in certain muscles to increasing stimuli. The response may be confined to certain muscles of the hip. It

is doubtful whether the plantar reflex is ever constantly and completely absent in healthy subjects. The term "flexor response" is proposed for this form of plantar reflex.

(3) In infants, up till the age of learning to walk, the succession of the contraction of the muscles in the plantar reflex is entirely different from that seen in the adult; the change from the one form of the plantar reflex to the other occurring gradually with the establishment of complete volitional control over the legs, usually between the second and third year of life. The term "infantile response" is proposed for this form of plantar reflex.

(4) During sleep the plantar reflexes are diminished, and the infantile and adult forms preserved, except in some children up to the age of 12 years, where in deep sleep the infantile form of reflex returns.

(5) In almost all cases of lesions of the pyramidal systems the form of plantar reflex is changed, a plantar reflex resembling closely that found in infants appearing. This change in the form of the reflex is one of the first signs of such lesions to appear, and is the last to disappear when the lesion is temporary. It may be the only unequivocal objective sign of a lesion of the pyramidal system. This form of reflex is never found under other conditions, and is a sign of great clinical value. The term "extensor response" is proposed for this form of the plantar reflex.

(6) In cases of total transverse lesion of the cord, the extensor response is the only reflex phenomenon present in the lower limbs.

(7) In functional cases the plantar reflex is often difficult to elicit, and there is frequently no response in the foot, but in such cases a response may usually be observed in the hip muscles. The form of reflex is the flexor response.

(8) In tabes dorsalis the plantar reflex was entirely absent in 20 per cent. of the cases. The response is often confined to the hip muscles. The attempt to obtain the reflex frequently may set up irregular movements of the foot lasting some time. The reflex when present is the flexor response.

(9) In peripheral neuritis the reflex when present is the flexor response. The hip muscles respond alone in some cases.

(10) In cerebral and cerebellar tumours the flexor response occurs if there be no involvement of the pyramidal system.

(11) In neurasthenia, chorea, paralysis agitans, poliomyelitis, myopathy and sciatica the flexor response is found.

(12) In lesions of the pyramidal system a degree of pes cavus is frequently found. It is intimately associated with the extensor response in the plantar reflex, and is produced by a state of reflex hypertonicity preponderating in those muscles which respond most vigorously in the plantar reflex. In all such cases of pes cavus evidence of increased tone in certain muscles can be demonstrated.

(13) The theory that the pes cavus of spastic conditions is due to weakness of the interossei is not justified.

REFERENCES.

[1] M. J. Babinski, *Le Semaine Médicale*, Juillet 23, 1898.

[2] Van Gehuchten, *Jour. de Neurologie Belge*, Avril 5, 1898.

[3] M. Brissaud, "Le Reflexe du fascia lata," *Gaz. hebd. de Med. Chir. Paris*, 1896, xliii., p. 253.

[4] Blocq and Onanoff, *Séméiologie et diagnostic des Maladies nerveuses*, p. 429, Paris, Masson, 1892.

[5] Gowers, "Manual of Nervous Diseases," vol. i., p. 14.

[6] Sherrington, *Lancet*, 1897, vol. i., p. 373.

[7] Bastian, "Paralysis Cerebral, Bulbar & Spinal," London, 1886, p. 580.

[8] Buzzard, "Clinical Lectures on Disease of the Nervous System," 1882, p. 118.

[9] Duchenne, *Physiologie des Mouvements*, p. 528.

[10] Golding Bird, *Guy's Hospital Reports*, London, 1885.

[11] Beevor, "Diseases of the Nervous System," London, 1898, p. 156,

[12] Adams, "Club Foot, its Causes, Pathology and Treatment," London, 1873, p. 94.

[13] Barwell, *Edin. Med. Journal*, 1898, N.S. 3, p. 113.

[14] Erichsen, "Text Book of Surgery," vol. ii., p. 530.

NOTES ON THE CHROMOPHILIC MATERIAL IN THE MOTOR CELLS OF BRAIN AND CORD, NORMAL (ANIMAL) AND PATHOLOGICAL (HUMAN), AND ON THE REACTION (ACID OR ALKALINE) OF THE CORTEX AND CEREBRO-SPINAL FLUID.

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I.

SINCE the publication in BRAIN (Winter, 1897) of a method of examining fresh cells, I have, at the suggestion of Dr. Mott, used a solution of methylene blue in normal salt.

With this the anterior cornual cells of the cow, pig, rat, rabbit and rook have been examined, as before described, as soon after killing as the cord could be extracted. In every case, even when no other medium (Farrant's solution) has been employed, and the stained little piece merely pressed out in its own moisture under a cover glass, the chromophilic elements have been distinctly visible in their usual form and arrangement.

And as regards human cortex no difference can be detected whether one uses a solution of methylene blue in water or in normal salt; and this refers to material which, in some cases, was examined only a few hours after death (from one to four or five hours).

These observations are in opposition to Held's view, that the form the chromophilic elements (Nissl bodies) assume is artificially produced by fixing reagents, but do not affect his further supposition that the acidity of the cortex set up after death, may precipitate the granules of

the hyaloplasm in the form of Nissl bodies. Van Lenhossek has seen in spinal ganglia cells brought under the microscope directly after death without any addition of reagents, the grains of the cell protoplasm.[1]

But although it may be shown that the Nissl bodies exist in quite fresh cells after death, there remains the possibility that they are precipitated as such by an acid reaction in the cortex, due to *post-mortem* changes. Held asserts that the Nissl bodies are insoluble in weak and concentrated mineral acids, but easily soluble in weak and concentrated alkalies, and he contends that during life the alkaline nature of the nerve cell fluid holds these bodies in solution, but that after death an acid reaction is set up by which a precipitation of granules takes place in the form of Nissl bodies.

There is a fact furnished by pathology and which will be further referred to later on, also opposed to this theory of Held's, and that is, that in certain morbid conditions, the bulky chromophilic spindles and blocks usually met with in healthy cells, dwindle away until they become the finest imaginable threads before disappearing altogether. The observation previously made that chromophilic material is met with in the fore-horn cells of animals freshly killed, does not prove anything decisively against the idea that the Nissl bodies are precipitated as granules from a fluid hyaloplasm that has become acid, because, however shortly after killing we test the nervous substance, we obtain both an acid and an alkaline reaction by using different indicators. This I have done with animals immediately after killing, and with human beings as short a time as seventy minutes after death, and the results of these experiments are given below.

Halliburton[2] states that he has never failed to find an alkaline reaction in fresh brain, cord or nerve, becoming, however, as a rule, rapidly acid after death. Langendorff (quoted by Halliburton) experimenting with frogs, rabbits

[1] Quoted by Goldschieder and Flatau. *Norm. und Path. anat. der Nervenzellen*, p. 14.

[2] Text book of Chem. Physiol. and Pathol., p. 515.

and guinea-pigs, finds the central nervous system to be alkaline during life, rapidly becoming acid after exposure or death.

Heidenhain and Gschleidlen state that the grey matter of the brain is acid even during life, probably due, they think, to lactic acid. J. Moleschott and A. Battistini, using phenolphthalein, found that the brain and spinal cord were acid, the grey more than the white.

With the exception of the last, no mention is made in Halliburton of the indicator used.

I have tested the reaction of the cortex and cerebro-spinal fluid in thirty-seven bodies of insane patients varying from one to forty-four hours after death, and using phenolphthalein, litmus, and methyl orange as indicators, and the grey matter (generally of spinal cord) of five animals (rook, rat, rabbit, pig, cow).

The results are always the same for the human cortex and cerebro-spinal fluid.

Phenolphthalein.—In all cases (twenty-seven human, and five animals) the grey matter discharged the pink colour from an alkaline (KHO) solution of the indicator.

The cerebro-spinal fluid of the human brains also *invariably* gave an acid reaction.

Litmus.—The paper was used; and when care was taken to avoid contaminating it by the cerebro-spinal fluid, the results were likewise constant.

In twenty-eight human brains the cortex was always acid.

Of the five animals, the rook's grey matter, tested one hour and forty minutes after killing, gave an acid. In the other four, tested as soon as the cord (or brain) could be extracted after killing, an amphoteric reaction was obtained, but the alkaline seemed the stronger, for when the blue paper was applied it turned faintly red; but this colour was evanescent, and faded after removal of the paper, but with red paper a permanent and decided blue was obtained.

With the cerebro-spinal fluid tested in thirty human brains, varying from seventy minutes to forty-four hours after death, there was also an amphoteric reaction in twenty-nine of the cases, for the blue paper was faintly tinged red, but

the colour quickly faded, whilst the red paper was somewhat slowly turned of a decided and permanent blue. In one case, five and a half hours after death, only an acid reaction was obtained.

Methyl orange.—This indicator, although it is not sensitive to most organic acids, reacts to lactic acid, yet, curiously enough, it only gives an alkaline reaction with cerebro-spinal fluid which after death contains this acid.

I have never obtained any reactions with yellow (alkaline) solutions, but with ten human brains and one cow's, the former varying from two and a half to twenty hours after death, a decided alkaline reaction always resulted.

With the cerebro-spinal fluid of sixteen cases also an alkaline reaction resulted in every one.

It will be seen from the foregoing that in one particular only does the reaction of the cortex differ from that of the cerebro-spinal fluid, in that the former is acid to litmus, the latter alkaline. Naturally the cerebro-spinal fluid, which permeates the cortex in all directions, will to a large extent determine its reaction; but the immediate and decided reddening of blue litmus paper shows that the grey matter contains substances not present in the cerebro-spinal fluid, or, if present, not in sufficient amount to influence its behaviour to litmus.

Lactic acid was tested for by carbol. ferric chloride (4 per cent. carbolic acid 10 c.c., water 20 c.c., liq. ferri perchlor. B.P. one drop) in sixteen cases.

A roughly quantitative estimation was attempted by always using 5 c.c. of the purple solution and counting the number of drops required to turn it completely yellow. This varied from seven to thirty, and, generally speaking, the number of drops was smaller the longer time which had elapsed after death when the fluid was tested, but not invariably. In three cases where the cerebro-spinal fluid taken from bodies seventy minutes, two and a half and twelve hours after death respectively was tested at the time of the autopsy and then allowed to stand for forty-eight hours, no very marked difference was found in the amount of lactic acid at these two different times, but if any reliance

is to be placed on the quantitative method employed, it indicated a slight diminution in the amount of lactic acid on standing.

Now, although lactic acid is found in the cerebro-spinal fluid as shortly as seventy minutes after death, it does not appear to be present in the fluid during life, nor to be formed in it after removal during life.

I have not directly tested for it under these circumstances, but from the fact that a sample removed by lumbar puncture in the case of a male general paralytic gave an *alkaline* reaction to phenolphthalein (which is sensitive to organic acids), I think it is highly probable that in this case lactic acid was not present. This same fluid, after standing forty-four and a half hours, still gave a distinctly alkaline reaction to phenolphthalein.

The above-mentioned observation tends to show that the lactic acid formed in the cerebro-spinal fluid after death is a *post-mortem* product, and that it is formed by chemical change taking place (*post-mortem*) in the brain; from whence it is carried to the fluid which bathes all parts of the brain.

I would note here that although with a freshly-killed cow a lactic acid reaction was slowly obtained from a piece of cortex in carbol. ferric chloride, yet this can quite readily be accounted for on the assumption that the protoplasm was killed by the reagent and produced the lactic acid in consequence there and then.

The results of these observations show that in human brains and animal both acid and alkaline substances are present in the cortex and cerebro-spinal fluid.

Certain German observers have already stated that such was the case, for Liebreich and Langendorff, using fresh pieces of cortex, found that they turned litmus blue, whilst Liebarkühn and Edinger, using an infusion of alizarin, got an acid reaction.

Summary.

(1) Chromophilic material in the form of Nissl bodies is met with in animals immediately after killing and in human

beings at least seventy minutes after death, when the nervous substance has merely been coloured by a solution of methylene blue in normal salt without any further addition of reagents.

(2) The fact that in some forms of diseased nerve cells the Nissl bodies show a gradual attenuation so that they dwindle down from bulky spindles and blocks to the finest imaginable threads, is opposed to the idea that they are produced by a precipitation after death.

(3) The reaction of the cortex varies with different indicators, showing that it contains both acid and alkaline substances.

(4) Its variations are constant, whether tested two hours or forty-four hours after death, and are as follows:—acid to phenolphthalein and litmus, alkaline to methyl orange.

(5) With animals killed and immediately tested, the grey matter gave similar reactions with phenolphthalein and methyl orange, but was alkaline to litmus.

(6) The reactions of the cerebro-spinal fluid differ from the cortex, in that with litmus an alkaline reaction is obtained. Otherwise they are the same. These results do not differ, whether the fluid is tested seventy minutes or forty-four hours after death.

(7) Lactic acid is present always in cerebro-spinal fluid after death, and appears to increase with the length of the interval which has elapsed between the time of death and the time of testing.

II.

Goldscheider and Flatau,[1] by subjecting rabbits to a high temperature in a thermostat, succeeded in producing a condition of pyrexia in these animals associated with a change in the fore-horn motor cells of the spinal cord. These changes were as follows:—

The cells appeared larger than normal, and stained (by Nissl's method) of a pale blue colour, opaque and homo-

[1] *Normale und path. Anat. der Nervenzellen.*

geneous; a finely granular condition could be discerned in relation with an ill-defined network of threads. Here and there were dark specks, probably the remains of Nissl bodies.

The nucleus was either not visible or indistinct, it stained homogeneously, and was sprinkled with the finest granules, and was not sharply outlined, and generally, as in the normal condition it was paler than the cell body.

The nucleolus was generally irregular, jagged, and darkly stained.

The dendrites were very pale and swollen, and presented slight varicosities; they showed no spindles, but only ill-defined granules.

As a result of numerous experiments, they came to the conclusion that a well-marked change of the above description was found as soon as the animal's temperature (which ranges normally from 101° to 102·7° F.) rose above 109·4°, and that if one allows the rise of temperature to last about three hours from 107·3° to 107·6°, then the first signs of alteration were visible on the periphery of individual cells.

Further, that a restitution from this condition occurred if the animal was allowed to recover; that it began after twenty-five and a half hours and was complete in sixty-eight hours.

Moxter, experimenting in somewhat different ways with artificially raised temperature in rabbits, obtained practically the same results as Goldscheider and Flatau, and he concluded that—

(1) A change in the forehorn cells is found after twenty-two and a half hours under the influence of a temperature between 104·9° and 106·7°.

(2) After several days' intermittent temperature between 100·4° and 106·3° no changes were found.

(3) Twenty-three hours, with a temperature varying from 102·5° to 105·2°, no changes were found, not even where the heightened temperature was several times renewed.

(4) That even in rabbits with lower temperatures than

those produced in the experiments an almost complete chromatolysis was met with, if the lower temperature was allowed to act sufficiently long.

Goldscheider and Flatau quote two cases where with pyrexia similar changes were found in the fore-horn cells of the cord. In the first—a man suffering from tetanus, whose temperature for the four days prior to death ranged from 98·6° to 101·1°, rising on the evening of death to 103·8°. The changes were found in all heights of the cord, in the cells both of fore and hind-horn, in the middle zone, and in Clarke's column. In the second—a girl with scarlet fever whose temperature for the last twelve hours varied between 104·9° and 105·6°—the cells of the cervical region were more affected than those of the lumbar.

Dejerine (quoted by Goldscheider and Flatau) found also in a case of pneumonia a decided chromatolysis of the nerve-cells of the spinal cord, which he considers similar to those seen in artificial pyrexia in rabbits.

Lugaro[1] asserts that experimental pyrexia causes complete disintegration of the chromatic part of the protoplasm of cortical and other nerve-cells without producing any other very marked change.

In a paper read before the Med. Psych. Soc. in May, 1898, I mentioned five cases of insanity in which I found changes in the motor cortex evidently very similar to those described (and figured) by Goldscheider and Flatau. At that time I was unaware of these experiments, yet the idea that they might have been caused by pyrexia had entered my mind, but had been dismissed because in other cases I had examined, where as high or higher temperatures had existed for some time before death, no such changes were found, and also because similar changes were occasionally met with in cases in which at least several days prior to death the temperature had not been raised.

Yet another reason against the view that these alterations were due solely to pyrexia was that in several cases where the altered cells were found in the cortex those of the fore-

[1] Quoted by Robertson and Orr. *Jour. Mental Science*, October, 1898, p. 740.

horn, cervical spinal cord, and hypoglossal nuclei showed a fairly normal arrangement and amount of chromophilic material.

Whilst I think that such reasons forbid us to regard pyrexia as the essential cause of the changes I described, it seems highly probable, considering how often pyrexia is associated with them, that such a condition will lead to alterations in the metabolism capable under favourable circumstances of producing these changes in the nerve-cells.

It is my intention here to consider in more detail the condition previously described, and to bring forward other examples since met with, because this condition seems to be of a very interesting nature, and also because others[1] have assumed that it was merely the accompaniment of pyrexia.

I do not for one moment wish it to be considered that it is peculiar to insanity—such is not the case; amongst the number now brought together is a case of typhoid fever in a female, whose brain showed the changed cells, and although she was not of normal intellect, her mental condition was such as to render it in the highest degree unlikely that the changes found in her nerve-cells were due to insanity, but that they were consequent on the typhoid fever.

Before proceeding to a description of the so-called pyrexial cell, a list is appended of cases in which there was a high temperature up to death and in which no trace of a single cell having any likeness to those in question was to be seen either in freshly squeezed-out films or in sections. A larger number of cases might have been brought together very easily, but I have restricted myself to those in which the temperature was ascertained to be high till at least a few hours before death so as to exclude the possible objection that restitution changes had taken place, and also to such cases in which beyond a certain amount of chromatolysis no very obvious alterations were noted in the cells when examined by the fresh method.

[1] Robertson and Orr. *Jour. of Mental Science*, October, 1898.

TABLE I.

No.	Sex.	Form of Insanity.	Age.	Cause of death.	Temperature in axilla (F.).
1	F.	Demented ..	33	Pneumonia	For two days prior to death varied between 101·2° and 102·8°, gradually rose till it reached 106° ten minutes before death
2	F.	Always weak-minded; maniacal	62	Abscess and necrosis of bone	For five days before death was always above 101°. (Highest = 103·8°.) On the morning of her death = 102°
3	F.	Chronic mania ..	48	Erysipelas ..	On the day before death it was 102° in the morning, 104° in evening. Not taken previously
4	F.	Puerperal mania	29	Pleurisy and Pneumonia	For the last few days as follows:—E., 106°; M., 103·8°; E., 105·2°; M., 103·6°; E., 105°; M., 103°.
5	F.	Acute mania ..	24	Pneumonia and Phthisis	For the last few days as follows: — M., 100°; 101·4°; 101·2°; 101·6°. E., 101·6°; 102°; 102·5°; 105°
6	M.	Idiot	7	Pneumonia and status epilepticus	The day before death as follows:—M., 105°; E., 103°. The next day it was 102·8° at 4 p.m., and he died at 9.25 p.m.
7	F.	Weak-minded; epileptic	27	Phthisis ..	For the last three days as follows: — M., 100°; 100·4°; 103·6°. E., 104·2°; 104·6°; 103°; falling to 101·6° at time of death.

If pyrexia alone could account for the change in cells under discussion, it is quite inexplicable why in such a case as No. 4, where for four days prior to death the temperature had stood at from 5° to 8° above normal, no such change was found.

Portions from the top of the ascending frontal of each of these cases were examined both in the fresh condition and after hardening.

(*A*) In the fresh cells merely coloured with methylene blue no very marked departure from the normal as regards the arrangement and quantity of chromophilic material could be determined.

In some the centre had a somewhat paler and washed-out appearance, whilst others showed granular degeneration of the Nissl bodies, but such were in a decided minority.

The cell bodies were generally irregularly angular, with many processes and not so large as in the so-called pyrexial cell, and the dendrites appeared normal in all respects, never showing the dull, pale blue uniform staining nor the very marked fragility found in such cells.

There were no special features which need be noted in regard to the nucleus. In some cases the nucleolus was large (12 μ) and pale, in others of normal size and dark. In others it had quite disappeared.

(*B*) In the portions of the ascending frontal which were fixed either in alcohol or sublimate, infiltrated with paraffin, and stained either with methylene blue, thionin or toluidine blue, there was a great uniformity in the appearance of the cells as regards the condition of the Nissl bodies (see figs. 1, 2, and 5).

Whilst many presented a fairly normal appearance, in the majority a very marked chromatolysis was found in all but one (No. 5) of the cases. Its characters were as follows:—

The apex, dendrites, and periphery of cell body always contained large, darkly-stained spindles, threads, or blocks.

The interior of the cell body is occupied by a paler and more uniformly stained substance, which resolves itself into a collection of pale and darker granules when highly magnified.

What I wish to draw attention to is the fact that although there is a considerable disappearance of chromophilic material in the cell, that which remains has not the appearance seen in the so-called pyrexial cell. There is no attenuation of the Nissl bodies; those which remain are as bulky and as deeply stained as usual. The condition of chromatolysis met with is quite unlike that obtained either in the partial disappearance of the Nissl bodies with pyrexia or their partial restitution with the subsidence of the pyrexia.

Except in one case (No. 7) there was no appearance of any undue shrinking of the cells, and their ground substance is nearly colourless.

The apex and dendrites are stout and have a regular outline presenting no varicosities. The nucleus is round or oval, generally with a distinct limiting boundary line, and more or less filled with a pale, amorphous material.

The nucleolus is generally darkly stained and regular in outline and varies from 4 to 6 μ in diameter. Often pale spaces or even vacuoles are seen in it.

Besides the ascending frontal in case No. 1 the frontal and occipital cortex regions were also examined, and in case No. 7 the cervical and lumbar enlargements of the cord and the hypoglossal nuclei. In the former case the pyramidal cells of the frontal region tallied with those of the ascending frontal, and a photograph of one of them is given in BRAIN (see Winter number, 1897, p. 450, fig. 1), which shows the large amount of chromophilic material and its arrangement. Similarly the giant-cells of the occipital cortex showed no marked deviations from the normal.

In case No. 7 both fresh and hardened specimens of cord were examined.

In fresh preparations of the hypoglossal nuclei the cells showed a perfectly normal appearance in the arrangement, &c., of the Nissl bodies. In the lumbar enlargement the motor cells of the fore-horn were fragile; the body of the cell was occupied by a mass of rather large granules of chromatin (granular degeneration). The dendrites appeared normal.

In sections.—Many of the fore-horn cells of the cervical enlargement presented considerable chromatolysis, characterised by the same features as mentioned in describing the ascending frontal cells, others appeared normal. In the lumbar enlargement a similar appearance of cells was seen as in the fresh preparations, the cell is filled with masses of chromatin rather smaller than usual and not rod-shaped (crumbling of chromophilic material). At the boundary the masses are larger and of a square contour. The dendrites appear normal.

Turning now to the cases which showed a similar condition of cells to that found in rabbits' cords after experimental pyrexia, the following table contains a list of these, and gives the form of insanity and the cause of death, &c.

TABLE II.

No.	Sex.	Age.	Form of Insanity.	Cause of death.	Remarks.
1	M.	40	Acute general paralysis	Pneumonia ..	Recently had a seizure before his death. Temperature not stated
2	M.	49	General paralysis	Seizure ..	His temperature rose to 105° F. before death
3	F.	35	Weak-minded from epilepsy	Status epilepticus; pneumonia	Temp. for last three days: M., 100°; 100·4°; 101°. E., 101·4°; 100·4°. Died 9.20 p.m.
4	F.	25	Acute puerperal mania	Peritonitis ..	Temp. at 6 p.m. of day of admission was 100·4°; next morning, 100·2°; 7 p.m., 103·4°; 9.15 p.m., 104·6°; 11.45 p.m., died
5	F.	49	General paralysis	Seizure ..	Temp. on morning of death = 103°
6	F.	58	Dementia and epileptic	Status epilepticus	Two days before death, evening temp., 99·4°; next morning, 98·4°; at 6 p.m., 106·6°. Died 7.30 a.m. next day
7	F.	24	Acute delirious mania	—	Temp. not raised for several days prior to death
8	M.	39	General paralysis (chronic lead poisoning)	General paralysis	Temp. not stated
9	F.	44	Weak-minded and fatuous; result of chronic alcoholism	Typhoid fever	Temp. high
10	F.	48	Chronic mania ..	Pneumonia and pericarditis	Had a temp. ranging between 102° and 104° for nine days prior to death
11	F.	40	Dementia ..	Cerebral tumour (gliosarcoma)	Temp. always subnormal.

Although in most there was a raised temperature immediately prior to death, yet there are two exceptions to this rule (Nos. 7 and 11), and in one other case (No. 3) it can scarcely be supposed that the temperature reached was sufficient, on Goldscheider and Flatau's showing, to have caused the changes in the cells which were met with.

Portions of brain, as in the last series, were taken from the top of the ascending frontal and examined both in a fresh state and after hardening.

(*A*) Fresh films stained in methylene blue.

Although the following description refers more particularly to the giant-cells of the cortex, yet it also applies to the other pyramidal cells. The cells appear large and blown out; they have to a large extent lost their multi-angular shape and have a more rounded contour. The cell body and apex stains of a dull, uniform blue, but with high magnification a finely granular condition is seen. Very often towards and in the apex are seen thin threads stained of a darker blue, which are the attenuated remains of Nissl bodies (fig. 7).

Although generally not much pigment is present, sometimes there is a large amount of it.

The dendrites, which are fairly numerous and fairly bulky, stain throughout of the same dull blue tint as the cell body. They are extremely fragile. The nucleus, when visible, is most often a pale, ill-defined zone around the nucleolus.

The nucleolus is generally central, and is very rarely displaced to the side of the cell. It is always round or oval, but varies considerably in size (from 5 to 12 μ) and depth of staining. To a large extent the latter is in inverse proportion to its size; the larger it gets the paler it becomes, until in some cells it is quite invisible.

In cases 2, 7, 9, and 10 it is in almost all cells very pale and large (11 μ), with a blurred outline. In some cells it is only just visible, in others quite invisible.

In cases 3, 5, and 6 it is generally small (5 to 6 μ), very dark—indeed, almost black—and stands out prominently from the pale background of the cell body.

In the other cases there is a more general mixture of the

two kinds described, so that it is difficult to say whether the large pale or the small dark nucleolus predominates.

The large pale nucleolus in sections from hardened tissue shows often as pale multi-loculated bodies not much larger than usual, the probability being that by dehydration they have shrunk.

Vacuolation or irregular staining of the nucleolus is not seen in fresh preparations before fixing.

With the exception of the differences noted in the nucleoli there is a great likeness between the cells of all the cases. My description will apply to the great majority of the cells seen, but some are met with which are very pale and appear as ghost-like forms of the others. In some of these pale cells a nucleus is seen staining of much the same depth as the cell body, with a definite but fine border.

(*B*) On examining sections which have been subjected to fixing agents (alcohol or sublimate) the cells no longer present in the different cases quite such an uniform character, but the difference is mainly in the shape and depth of staining.

The giant-cells are generally large and rounded, filling out the entire pericellular space, but with some cells there appears to have been considerable shrinking, and this space is large.

The dendrites are manifestly thinner than in healthy cells, and probably less numerous, and not infrequently show varicose dilatations.

Sections stained in thionin or toluidin blue present a very characteristic appearance, owing to the universal but pale violet-tinted staining of the cell and its processes. The whole of the protoplasm staining of the same depth. It appears impossible with these stains to get a deep colour in the cells. High magnification of the apparently uniform cell body shows a delicately granular background, with larger and darker granules sprinkled about. In many cells remains of Nissl bodies can be detected in the apex and about the periphery. These are in the form of delicate threads (figs. 3, 4, 10, and 11).

This gradual attenuation of the Nissl bodies, until the

originally big spindles or rods have dwindled away to fine threads, is a point worthy of note in reference to the question as to the origin of these bodies by precipitation, and cannot easily be accounted for by Held's theory.

In the sections a large cell is occasionally seen which has absorbed the stain deeply, and appears blotchy, showing little or no detail, but these are in most cases very few and far between.

The nucleus presents no peculiarities from those described before; we see an ill-defined granular mass arranged more or less in the form of an irregular mesh work, and described as such by German observers, but which it seems more likely is merely the representation of a coagulated albuminous mass, which during life and before fixing occupied this body as a fluid or semi-fluid material.

The nucleolus varies somewhat, corresponding probably to the variations noted in the films. It often, although darkly stained, shows a number of pale or colourless spots (fig. 4). In other cases it is pale and with a multiloculated appearance.

There is not the extreme difference in size met with in the fresh preparations, probably because the large pale nucleoli noted in the latter are more fluid in nature and have contracted more when subjected to dehydrating agents.

In cases 7, 10, and 11, besides the ascending frontal, other parts of the nervous system were examined, viz., the cervical cord in No. 7 and cervical cord and hypoglossal nuclei in Nos. 10 and 11.

In these parts, both in fresh and hardened preparations, the chromophilic material appeared generally in normal amount and arrangement, but in some few cells chromatolysis of the character previously noted was seen.

There can be no question that as far as morphological appearances go the cells of all the cases, with the exception perhaps of one (No. 7), represent a similar pathological condition, and as regards this one there are certain facts which render it probable that it belongs to the same category, but probably in a more advanced state of disease.

In fresh preparations its cells appeared identical with

those of the other cases, and in certain parts of the sections, always at the base of a convolution, some were seen which were not shrunken and appeared identical to those seen in sections from the other cases, also in nearly all the sections from these latter certain cells were met with which stained densely and appeared shrunken, and, in fact, had very much the appearance of the majority seen in No. 7.

Case No. 7 appears of sufficient interest to warrant a brief description. It was that of a well-developed, strong girl, aged 21, who, previously to her mental affection, had always had good health. She was admitted here suffering from acute delirious mania of only a few days' duration, and whilst in this state had jumped out of a bedroom window and badly bruised herself, but beyond the bruises did not appear to have sustained any serious injury to her system.

She was violently excited for the first few days after admission, and then showed signs of collapse with intervals of restlessness, when she would toss her arms about and move her head. A large bleb formed on her right heel, her tongue, which at first had been heavily furred but moist, became dry, brown, and cracked, and she died seven days after admission. Her temperature and pulse was as follows: —August 2, 5.30 p.m., 101·4°; pulse 123. August 4, 4 p.m., 97·8°. August 5, 6.45 p.m., 99·8°; pulse 142. August 6, 9 a.m., 98·6°; pulse 123; 7 p.m., 99·2°. August 7, 7.30 a.m., 98·6°; pulse 124; 7 p.m., 98·6°. August 8, 8.30 a.m., 98·6°; 6 p.m., 98·4°. August 9, 8.30 a.m., 98·4°; 7.30 p.m., 98·2°. She died at 6.20 a.m. on the 10th. The autopsy was held four and a half hours after death.

On the external surface of the dura mater was a thin, soft, dark clot, 40 × 30 mm.; it lay over the middle portion of the right second frontal convolution. The veins of the arachnoid were much engorged. There was excess of pale, reddish fluid in the lateral ventricles. No other morbid appearances noted in the head except perhaps some shrinking of the convolutions at the vertex, and the cortex was dark in colour on section. The encephalon weighed 1,218 grammes. The heart was small (245 grammes), and there were some atheromatous patches on the aorta near the valves. The

liver was pale. In other respects the thoracic and abdominal viscera appeared healthy. Portions of the ascending frontal at the top on both sides were examined fresh, other pieces from the same region were fixed in 10 per cent. formal. followed by absolute alcohol. Pieces of the liver and kidneys were put into Müller, and when examined microscopically the latter, except for very slight fatty degeneration in the superficial parts of the cortex, appeared fairly normal, but the liver showed very general and marked fine, fatty degeneration at the centre of the lobules.

In the brain the fresh films showed the giant-cells of the motor cortex, quite indistinguishable from those already described in general appearance and with the same extremely fragile dendrites. Fig. 7 shows one of them. There is evidence of remains of Nissl bodies in the form of fine threads in the apex.

The nucleolus is large, pale, and indistinct. The dendrites stain of a pale, dull blue, and all over alike.

The only difference noted in the cells was that some were pale and ghost-like, others darker and more dense.

In the sections the nerve-cells of the second layer appeared normal, but in all the deeper layers they were much altered. The pyramidal cells staining uniformly throughout of a somewhat paler colour than usual and with absolutely no chromophilic material, presented identically the same appearance as the pyramidal cells seen in the other cases of Table II.

The greater number of the large nerve-cells (giants) were much shrunken, darkly stained, and somewhat distorted, with a wide pericellular space around them. The staining of the cell body was so dense and diffuse that all detail was obscured (see figs. 6 and 8).

The nucleus was generally central and round or oval, but sometimes irregularly encroached upon by the protoplasm of the cell body. Nucleolus, round and dark. The dendrites, few in number, were dark and thin and devoid of chromophilic material, showing in places varicose dilatations along their course. The apex was very irregular in contour and in the way it stained, being very dark in some places, pale in others.

Others of the big nerve-cells were less darkly stained, and appeared as though consisting of fibres arranged as an irregular meshwork, whilst the apex had a rough, unravelled aspect (fig. 9). And lastly, as before mentioned, a few cells characteristic of the other cases of Table II. were seen.

The blood-vessels were tortuous and showed masses of pigment, staining of a green colour, lying in the adventitia and perivascular spaces. In some of the larger vessels in the pia there was an aggregation of numbers of polymorphonuclear leucocytes filling up their lumen, whilst others were wholely or partially blocked up by a homogeneous hyaline material which stained of a greenish blue.

No appearance of any neuroglia proliferation.

The upper part of the cervical cord was also examined. The fore-horn cells were shrunken and deeply stained; the majority show the usual arrangement and quantity of Nissl bodies, whilst others with their dendrites are throughout stained darkly.

Now, unless we suppose that the fall, which probably was the cause of the small hæmorrhage on the dura, also produced such disturbance in the cerebral hemispheres as to give rise to the condition of cells found—a supposition not very probable—then we must take it that the pathological appearances noted were directly associated with the insanity.

What relation did the one bear to the other?

I would suggest from the following considerations:—

(1) The acute nature of the mania and its rapidly fatal result,

(2) The absence of naked eye lesions to account for such a termination,

(3) The changes in the nerve-cells, and

(4) The highly suggestive fact of the fatty degeneration of the liver in so young a girl.

That a wide-spread toxic influence was at work and that the chain of events was somewhat as follows:—

(1) Mental disturbance.

(2) Perverted metabolism resulting in the setting free of toxins in the system.

(3) Action of these on already disordered nervous system.

This also is the hypothesis that I would advance to account for the changes in the cells of the other nine cases.

A glance at the cause of death in Table II. will at least show that the circumstances under which we come across such cells are highly suggestive of poisoning, and to give an instance illustrating this we can almost count upon meeting with them in the brains of general paralytics who have died in seizures.

But whether this explanation is accepted or not, I think that sufficient reasons have been brought forward to show that the changes under consideration are not due directly or solely to pyrexia.

Summary (Part II.).

Nerve-cells are met with in the human cortex, which are similar in appearance to the fore-horn cells found in the cords of rabbits after experimental pyrexia, but that pyrexia alone cannot account for their condition is shown by the following facts:—

(*a*) The frequent occurrence of pyrexia lasting until death in cases where such cells are not found, either in brain, hypoglossal nuclei, or cord.

(*b*) The occurrence of these cells in the cortex of cases which had shown no pyrexia, or in which the pyrexia had been too slight to account for them.

(*c*) And finally, the fact that in certain cases, while these cells were found in the cortex they were not met with in the hypoglossal nuclei or in the cervical cord, the nerve-cells in these places showing the presence of Nissl bodies in a fairly normal condition.

Note.

Since the above was written I have examined the brain from a man, age 34, suffering from acute mania, who died of diphtheria. His temperature two days before he died was 102° F., and although I have no record of it before that time, there can be no reasonable doubt that it was high for some

days previously, as when it was first taken the disease was in an advanced stage and his neck glands much involved. The day following it had risen considerably, and shortly before his death was 104° F.

Portions of the ascending frontal convolutions of both sides were examined fresh and after fixing in sublimate.

As regards the fresh method, although some eight or nine films were prepared and numbers of large nerve-cells seen in all, not one showed any appearance of the so-called pyrexial cell.

A very considerable number was stained of a dark, bright blue and had a normal stichochrome arrangement of their chromophilic elements throughout the entire cell; the dendrites were bulky and unstained, except for distinct threads of chromophilic material.

The majority, whilst presenting a similar appearance to the above in apex, dendrites, and periphery of cell body, showed in the interior a condition of granular degeneration of the Nissl bodies.

In some the granules are big and close set (dark cells), in others smaller and further apart (pale cells).

A perfectly analagous appearance of big cells was seen in the sections; and, in respect to the smaller pyramidal cells, although the chromophilic material may have been diminished, yet they failed entirely to show the quite characteristic appearances described before in the cases associated with the so-called pyrexial cells.

In many of the big cells in the films the nucleolus was pale, ill-defined, and more or less increased in size, in others it was quite invisible; but in the sections all of those seen were dark, sharply defined, and not above their usual size.

That there is some relation between certain fevers and states of insanity has long been recognised, and it is interesting to note that manifest changes of the nerve-cells can be detected in two of those fevers after attacks of which improvement or recovery of mental condition has occurred. I refer to the typhoid fever case recorded above and to a case of scarlet fever mentioned by Goldscheider and Flatau. In this case, however, only the changed condition of the

motor cells of the spinal cord is referred to, and apparently the brain was not examined.

As far as I know no such improvement has been noted in cases of insanity after attacks of diphtheria, which does not appear to affect the central nerve-cells so profoundly or in which at least there is not the very palpable alteration such as are seen with typhoid and scarlet fever.

DESCRIPTION OF THE FIGURES.

All were photographed with $\frac{1}{12}$ oil immersion (Beck), No. 2 eye-piece ($=\frac{2}{1}\frac{0}{0}$), with the exception of No. 7, which is with $\frac{1}{6}$ in. This last is from a fresh preparation, the others from sections.

FIG. 1.

Shows a fairly normal arrangement of Nissl bodies in the form of blocks and granules; the axis cylinder is also seen, and one stout dentrite with chromophilic threads in it (Case 5, Table I.).

FIG. 2.

Two cells from same case with normal arrangement of Nissl bodies. At the lower left hand part of the right cell is the axis cylinder eminence. Some pigment at the lower end of left cell.

FIG. 3.

Two cells from Case 10, Table II. Both stain diffusely and rather deeply, and show coarser granules on a finer granular background. In the upper cell a few attenuated threads of chromalin are seen in the apex. Small vacuoles in nucleolus.

FIG. 4.

Paler cell from same case with pigment at lower end. The attenuated chromophilic threads are well shown. Nucleolus vacuolated.

FIG. 5.

Cell from Case 3, Table I. Pigment in lower end. Bulky chromophilic spindles in upper part. Nucleolus vacuolated. A stout dendrite shown, unstained except for the chromophilic threads.

FIG. 6.

A darkly stained cell from Case 7, Table II., showing no chromophilic material. Apex and dendrites darkly stained throughout; the latter thin, and with varicose dilatation.

FIG. 7.

Same case. A fresh cell with a small patch of pigment at lower right end. Faint remains of chromophilic material seen in apex. Dendrites stain uniformly and are devoid of chromophilic material. Nucleolus ill-defined.

FIG. 8.

Same case. Darkly stained cell, showing a very ragged and irregularly contoured apex.

FIG. 9.

Same case. Pale cell with rough, irregular mesh work appearance of body and apex.

FIGS. 10, 11, 12.

Cells from a case of typhoid fever, Case 9, Table II.

Fig. 10 is a rather darkly stained cell with thin threads of chromophilic material shown in the apex end processes. Nucleolus is vacuolated. Fig. 11 also shows fine threads in apex and upper two-thirds of body; the lower third is occupied by pigment. Fig. 12 is a very pale cell, uniformly stained, except for one or two little masses of chromophilic material. Nucleolus is vacuolated.

FIG. 1.

FIG. 2.

FIG. 3.

FIG. 4.

FIG. 5.

FIG. 6.

FIG. 7.

FIG. 8.

FIG. 9.

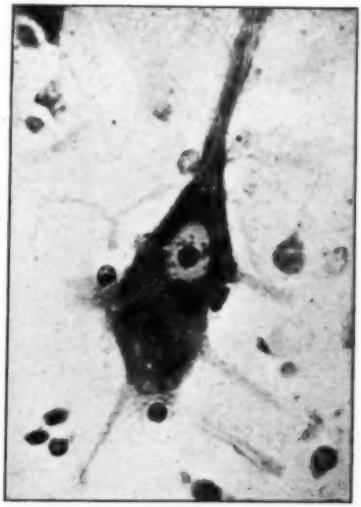

FIG. 10.

FIG. 11.

FIG. 12.

ON A FORM OF NERVE TERMINATION IN THE CENTRAL NERVOUS SYSTEM, DEMONSTRATED BY METHYLENE BLUE.*

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SINCE the introduction of the chrome-silver method into nerve histology, our hitherto accepted views upon the relations of nerve-cells have been completely transformed.

The two chief conclusions based upon this method are: first, that axis-cylinder processes and their collaterals, as well as protoplasmic processes, have free terminations; and secondly, that there is no such thing as anatomical continuity of nerve elements.

In 1891 Waldeyer[1] introduced the term *neuron*, which he defined as follows:—"That the central nervous system is made up of numerous nervous units; that the axis-cylinder in every case springs directly from the cell, and further, that all nerve-fibres or fibrils have free terminations by means of tufts or brushes (endbäumchen, endbuscheln). There is no such thing as formation of net or anastomosis."

The first light thrown upon the cellular structure of the central nervous apparatus was in 1865, when Deiters[2] pointed out the distinction between axis-cylinder and protoplasmic processes. His results were soon confirmed by many observers, as Arndt,[3] Schultze,[4] Kölliker,[5] Retzius,[6] and others.

Meynert[7] and Besser[8] believed that the protoplasmic processes of different nerve-cells anastomosed freely with

* We are much indebted to Professor Ferrier for the opportunities afforded to us for this investigation.

each other. In 1871 Gerlach[9] propounded the theory that a network was formed, composed of afferent fibres and the protoplasmic processes of nerve-cells, out of which arose axis-cylinders. That is to say, there was no real difference between protoplasmic and axis-cylinder processes and the grey matter was a network of continuous strands.

The question may be said to have been left open until the publication by Golgi,[10] in 1875, of his method for impregnating the brain with silver. The conclusions which he deduced were, first, that the network was made up only of axis-cylinder processes and collaterals; and secondly, that the protoplasmic processes were to be regarded as subserving nutrition and had relations with the blood-vessels and ependyma cells.

In 1887 Forel,[11] Retzius[12] (using Ehrlich's original methylene blue method),* and His[13] (by embryological research), were led to doubt the existence of the network, believing in the discontinuity of the elements.

Later, the researches of Ramon y Cajal,[14] Kölliker,[15] van Gehuchten,[16] and Lenhossék[17] confirmed their results, that the grey matter was composed of a feltwork of interlacing but discontinuous fibrils.

In this way arose the present accepted theory of transmission of impulses by contact only.

It would appear, however, as if the varieties of nerve termination, described by those who have largely employed the chrome-silver method, are not the only forms existing in the central nervous apparatus. There is also reason to doubt whether the terminations, described by these observers, are so universal as the study of their writings would lead one to suppose. In 1886 Ehrlich[18] initiated the method of staining nervous tissue by methylene blue. Owing to the exceptional position which Golgi's impregnation method attained, years passed before Ehrlich's method came into anything like general use. Interest in the subject, however, was never

* Retzius, in invertebrates, injected 0·2 per cent. solution of methylene blue into the body. After a few minutes the central organs were laid bare and exposed to the air. Examination was made from time to time in order to determine the time required for good results. The specimens required to be examined microscopically at once.

entirely lost, and from the first papers on the subject by Aronsohn,[19] until the present day, it has been more and more recognised that in this one has to deal with a method as original as, and perhaps superior to, the impregnation methods of Golgi and Cajal. In fact, the method, if carried out successfully, bears out Ehrlich's original statement: "*That methylene blue possesses an extraordinary relationship to the finest and most delicate branches of axis-cylinder processes, and therefore it is possible to follow out definite nerve endings in the living condition and with a clearness that cannot be attained by any other method.*"

In 1895 Bethe[20] discovered that by the use of certain salts—the molybdates—methylene blue could be fixed in the parts already stained. This made it possible to obtain good sections from parts selected for examination. His researches were, however, mostly confined to invertebrate animals. Shortly after this Semi Meyer[21] applied Ehrlich's method to vertebrate animals by means of subcutaneous injections of methylene blue, employing as his fixing agent one of the solutions recommended by Bethe. By this means the brains of even the higher mammals can be thoroughly saturated with methylene blue, afterwards fixed, embedded, and sectioned. If this be carried out carefully and successfully, the structure of the central nervous apparatus is brought out with a clearness that cannot even be attained by the use of the chrome-silver method.

Meyer,[22] in his latest publication, describes a variety of nerve termination which he has met with in various parts of the brain, and comes to the conclusion that it takes the form of a very fine basket of fibrillæ over the cell body and protoplasmic processes. From his preparations, however, no conclusion could be drawn whether this basket-work was made up of a meshwork of continuous fibrils, or of a texture in which each fibril retained its individuality.

He further points out the relations of the dendrites to the cellulipetal fibre. According to Cajal and v. Gehuchten, the protoplasmic processes are the parts of the neuron which receive afferent impulses and conduct them to the cell. Meyer's preparations have led him to believe that there

exists no difference between the cell body and protoplasmic processes, the basket-work being as clearly defined on the dendrites as on the cell body, and that the afferent fibre in most instances is found to enter into relation with the neuron near the summit of one of its protoplasmic processes.

This type of termination has been recently noted by Auerbach,[23] Bethe,[24] Golgi,[25] and Held,[26] but from the descriptions given, one is unable to form an idea as to the ultimate behaviour and relation of the cellulipetal fibre to the body and protoplasmic processes of the cell around which it ends.

The use of the chrome-silver method and its modifications is open to objection. In fact, the number of modifications, which it has already undergone, clearly prove the imperfections inherent in it. Being a precipitation method it obscures structure; that is to say, the finer structural details of the elements impregnated cannot be investigated owing to the intensely black deposit on the cells and branches proceeding from them.

The formation of artefacts, the capriciousness of the method, its imperfect durability, and lastly, the defective means it possesses of differentiating nerve-cells from neuroglia cells, constitute additional imperfections. Therefore the use of other methods is necessitated in order to examine in detail the structure of the tissue elements.

It has been noted that most of the imperfections mentioned can be overcome by the use of subcutaneous injections of methylene blue *intra vitam*. This method is much more certain and the structure of nerve-cells, processes, and nuclei can be made out. By the use of methylene blue neuroglia elements are eliminated, nervous tissues only being stained, and medullated as well as non-medullated fibres are defined.

Our Own Observations.

The method which has been adopted by us is briefly as follows:—Small quantities of a saturated solution of methylene blue are injected subcutaneously at intervals of from

fifteen to twenty minutes until the animal dies. The central nervous organs are rapidly removed, sliced into convenient blocks, and placed in a solution of ammonium molybdate for purposes of fixation. After washing, hardening, and embedding, the blocks are cut as usual and mounted with a cover-glass. Various animals have been employed for this purpose—monkeys, cats, dogs, rabbits, guinea-pigs, and mice. The best results are obtained with rabbits from six weeks to two months old.

Description of the Nerve Termination.—The arrangement which we regard as the termination of a nerve fibre is in the form of a lattice or network surrounding chiefly the cell body. For the sake of clearness this may be described as studied under different powers of the microscope.

(I.) Under $\frac{1}{6}$ inch objective (Swift), and the nucleus of the posterior quadrigeminal body selected for preference.

The terminal is to be found in connection with the large cells of this nucleus, and the following appearances would indicate that we have to deal with a pericellular arrangement (plates i. and ii., figs. 1, 4 and 5).

(*a*) When in clear focus the cell shows a deeply blue-stained peripheral zone.

(*b*) When thrown out of focus the characteristic cell appearance is replaced by a series of fibrillæ radiating over the cell and nuclear region, in connection with which are numerous deeply-stained particles. This is not clearly portrayed until the nucleus is almost invisible.

(*c*) A similar condition may be observed over the roots and main stems of the protoplasmic processes.

(II.) In order to define more clearly the minute structure it is necessary to study the cell under $\frac{1}{12}$ inch oil immersion (Swift), when the following may be seen:—

(*a*) The peripheral zone is found to be nodose and conterminous with the periphery of the cell. At times it presents almost a "thorny" appearance (plate i., fig. 2). When the cell is in clear focus the peripheral zone is found in almost all cases to cross the base of the protoplasmic processes.

(*b*) When the cell is thrown out of focus the fibrillated structure already mentioned is now found to be a lattice work enshrouding the cell (plate i., figs. 1 and 5).

The particles, to which reference has already been made, represent the points of interlacement of the fibrillæ. The general appearance is that of a very fine web or meshwork covering the cell and roots of the protoplasmic processes. In favourably stained specimens, there is one part of the cell in which the lattice work is most clearly seen, namely, over the root of the axis-cylinder process, where, as clearly shown by other methods, there is more or less deficiency in the so-called "Nissl bodies." In these cases the deeply-stained network stands out clearly from the faintly-stained cell background.

The Cellulipetal Fibre.—As far as we have been able to observe, this fibril is characterised by the following appearances (plate i., fig. 3):—

(1) It is a fine, deeply-stained fibre.

(2) On various parts of its course it presents a looped or varicose arrangement, occurring either singly or in pairs, the latter having a characteristic "pair of spectacle" appearance.

(3) From it collaterals are given off at right angles.

(4) It does not present a protoplasmic structure like dendrites.

(5) It never presents a thorny appearance.

(6) In some cases it reaches the cell body by way of the principal stem of a protoplasmic process (plate ii., fig. 3).

(7) There is no evidence of its breaking up into a network before reaching the cell body.

Localities in which the Termination has been found.

(1) *Cerebral Cortex* (plate i., figs. 1 and 2).—According to our experience, nerve-cells take on the methylene blue stain in two ways, whether in the cerebral cortex or in other parts. In all regions which we have examined, deeply as well as faintly-stained cells have been observed.

It is in connection with the faintly-stained cells that we find the terminal arrangement most typically developed; but it is also to be seen in relation with those which take on the stain more deeply.

In successful preparations, numerous examples of the lattice work are to be found in all areas of the cortex, but

more especially in that corresponding to the Rolandic area in man. They are limited, so far as we can make out, to the large and small pyramidal cell layers, but there is no clear evidence as to which cells the ending stands in relation. The cellulipetal fibre appears in most cases to run in from the molecular layer and meet the cell near the base of a principal protoplasmic process. We are unable to confirm Meyer's conclusion that the afferent fibre splits up on a protoplasmic process near its summit.

(2) *Optic Thalamus.*—One of the best examples of this type of termination was found in this ganglion (plate ii., fig. 3). The ending was in relation to a large, three-cornered cell of the pulvinar. The cellulipetal fibre joined one of the principal protoplasmic processes at some distance from the body of the cell, but did not break up into a lattice work until it reached the cell body. Before reaching the protoplasmic process this fibre showed distinct thickenings or nodes at irregular intervals.

(3) *Internal Geniculate Body.*

(4) *Anterior and Posterior Quadrigeminal Bodies* (plate i., figs. 4 and 5).

(5) *Red Nucleus.*

(6) *Lateral Fillet Nucleus.*

(7) *Superior Olivary Body.*

(8) *Ventral Auditory Ganglion.*

(9) *Tuberculum Acusticum.*

(10) *Deiters' Nucleus.*

(11) *Dorsal Vago-glossopharyngeal Nucleus.*

(12) *In Motor Cranial Nuclei, namely, Oculo-Motor, Facial, and Hypoglossal.*

In the above-mentioned motor nuclei we have found the endings always in connection with faintly-stained cells, which present characters as regards shape, number, and length of protoplasmic processes, &c., differing from those which take on the stain more deeply, namely, multipolar cells. Meyer has observed a similar condition, and it would appear from our preparations that at least two varieties of nerve-cell are present in those nuclei.

Thus far we have been unable to find this termination

in the substantia gelatinosa of Rolando, or in any part of the spinal cord. These are parts of the central nervous system in which, according to the chrome-silver method, so-called nerve endings are specially well seen (Cajal, Kölliker). To account for this discrepancy we are unable at present to offer any explanation.

(13) *Cerebellum.*—Apart from the corpus dentatum cerebelli, no termination of this nature has been discovered. In this latter nucleus the lattice work, surrounding the cell body and principal protoplasmic processes, presents the same characteristics as elsewhere.

Not only does the methylene blue method fail to demonstrate terminations, as we have described, in connection with Purkinje's cells, but there is an entire absence of the structures known as "the pericellular basket-work" and the "fibres grimpantes," described by Ramon y Cajal. As the staining property of methylene blue is purely confined to nervous elements, neuroglia and connective tissue being unaffected, we would argue that these arrangements are of a non-nervous nature.

As is well known, by the use of the chrome-silver method, the formation of artefacts, namely, the spread of deposit, the staining of the lymph case, and the degree of impregnation depending upon the texture of the tissue, is of frequent occurrence in the cerebellum. We are, therefore, inclined to believe with Hill[27] that the pericellular baskets and "Kletterfasern" surrounding Purkinje's cells and processes are not the end terminations of nerve fibrils.

Further, the "fibras musgosas" (mossy fibres) are shown to great advantage by means of this *intra-vitam* method, the thickenings or rosettes being very clear in outline in well-stained preparations.

In no case, however, have we been able to demonstrate endings around the granule cells, as described by Dogiel, nor can we support the hypothesis of Cajal, that the dendrites of the granule cells break up over the bodies of their neighbours.

(14) *Nucleus of the Corpus Trapezoides* (plate i., fig. 3).—This nucleus, described by Held[28] and Kölliker[29], occupies a position in the ventral part of the tegmentum, between the superior olivary body and the roots of the abducens.

Kölliker, with the chrome-silver method, described the cells of this nucleus as being round or spindle-shaped, and possessing axis-cylinders which proceeded from the cell body by means of a number of small, thick branches, fusing at some distance to form the efferent fibre. At first he declared this to be an artefact, but in his later publications regards only the thickness of the fibres to be due to artificial production. That this appearance was not the commencement of axis-cylinder processes, but really the end termination of nerve-fibrils, was first pointed out by Held. Later, the researches of Cajal[30] and Meyer[31] have placed the matter beyond dispute.

According to Meyer, these afferent fibres, just before ending, become gradually thickened, and near the cell break up into a variable number of branches, which clasp the cell, forming a basket or shut cup. Cajal, however, has come to the conclusion that no basket is formed, but the ending is to be regarded as an open cup, or, as he terms them, "löffel formige plaques."

Our own observations tend to confirm Meyer's conclusions, namely, that it is the most easily demonstrable end termination in the central nervous system; that it takes the form of a network enveloping the cell body, and, finally, in opposition to Held, that there is no direct continuity between the termination and the protoplasm of the enshrouded cell.

Conclusion.

For the following reasons we would urge our belief that the above described arrangement is really the termination of a nerve fibre:

(1) That in the majority of cases a cellulipetal fibre may be traced to, and found to break up over, the cell body and base of the protoplasmic processes. In no case has the cellulipetal fibre been found to break up previous to reaching the cell body.

(2) That this lattice-work is most clearly defined over that portion of the cell, namely, the root of the axis-cylinder process, which is deficient in the so-called Nissl bodies.

(3) That it is also clearly seen over cells whose general staining is faint.

(4) That when the cell is rendered indistinct by changes in the focus, the lattice-work is thrown into clearer perspective.

(5) That, as best seen in the nucleus of the trapezium, the ending may be apparent without the cell.

Remarks.

The variety of nerve-termination just described is in complete harmony with the neuronic theory of nerve elements, at present holding the field in nerve anatomy; that is to say, the neuron is a unit, discontinuous with adjacent neurons, and conveying impressions by contact and not continuity of tissue. This termination, demonstrated by means of *intra-vitam* injections of methylene blue, is in the form of a fine lattice-work, forming the extremity of the axis-cylinder process of one neuron, enshrouding the body of the neuron next in order. Although physiologically more or less in harmony, this is really anatomically at variance with the descriptions given by Golgi, Cajal, and others, of the ultimate termination of nerve-fibrils. These observers have worked chiefly with the chrome-silver method, which, being a *post-mortem* impregnation process, obscures structure and does not allow the finer details to be investigated.

With the exception of the pericellular basket-work and "Kletterfasern" around Purkinje's cells in the cerebellum, to which further reference will presently be made, the generally accepted doctrine is as follows: first, that the "terminal" consists of a tuft of nerve-fibrils whose extremities may be in some pointed, in others knob-like; secondly, that in the meshwork in the grey matter, which is thus formed, these come into contact with the arborisations of the protoplasmic processes of cells; and thirdly, that nerve impulses are transmitted from the terminal tuft along these protoplasmic processes to the cell body, and thence to the axis-cylinder. Our conception of the method of transmission of impulses from one neuron to another, in view of what we have observed, is a modification of that currently held. From the fact that the cellulipetal fibre breaks up into a network in relation with the cell body, and that the mesh

cannot be traced for more than a short distance over the chief trunks of the protoplasmic processes, we assume that the impulses are transmitted from the terminal directly to the cell body and thence to the axis-cylinder.

What the function of the protoplasmic processes may be is less clear. Golgi traced the protoplasmic processes into connection with the blood-vessels and ependyma cells, and regarded them as subserving nutrition. Cajal and v. Gehuchten believe that they alone are the chief collectors of impulses and conduct them to the cell body.

According to S. Meyer, who has worked with the methylene blue method, the terminal arrangement is found over the whole extent of the protoplasmic processes and cell body. He therefore concludes that there exists no real difference between the cell body and protoplasmic processes, a view which receives support from a study of ganglion cells, after staining by the Nissl method, by which it is seen that the chromatic granules are found in the protoplasmic processes and cell body, but not in the axis-cylinder.

From our own observations, however, we would incline to the old view of Golgi, that their chief function is nutritional, or, at all events, of a different functional nature to the axis-cylinder process. In support of this theory, Bethe and others have recently demonstrated by other methods that certainly the protoplasmic processes and perhaps the nucleus and protoplasmic accumulations of the ganglion cell subserve nutrition.

As already incidentally mentioned, the only structures comparable to what we have described, detected by the Golgi-Cajal method, are the so-called "basket arrangements" round the Purkinje cells in the cerebellar cortex. Cells are described, situated in the molecular layer, whose axis-cylinder processes and collaterals end in a brush of fibrils, which form a basket investing the body of a Purkinjé cell. As the end filaments approach these cells, each of them divides into a brush or basket, in which, however, each fibre, according to Cajal and v. Gehuchten, retains its individuality. From the fact that there is no evidence of these structures on *intra-vitam* staining with methylene

blue, and also that they do not form a lattice-work of continuous fibrils in any way comparable to that which we have described in other parts, we do not regard them as of a nervous nature, but neuroglial. Hill's conclusions as to the drawbacks to the chrome silver method, namely, spread of deposit, staining of the lymph case, and the open nature of the tissue around Purkinje cells, together with the fact that by the use of methylene blue neuroglia elements are eliminated, strengthen our belief as to the neuroglial nature of the basket arrangements and "Kletterfasern" around Purkinje's cells.

In conclusion, control observations by means of *post-mortem* methods have failed to demonstrate this lattice or network termination. It would seem necessary that, in order to obtain any idea as to the ultimate subdivisions of nerve-fibrils, the tissues should be given the opportunity of selecting the stain during life.

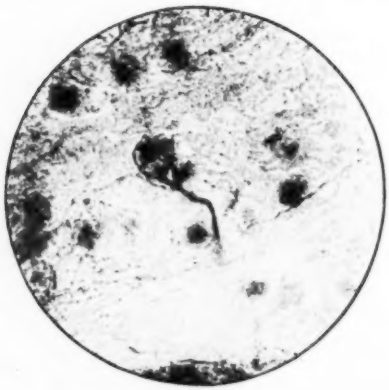
LITERATURE REFERENCES.

[1] WALDEYER, *Deutsch. Med. Wochenschr.*, No. 50, 1891.
[2] DEITERS, *Untersuch. über Gehirn und Rückenmark des Menschen und der Säugethiere; Braunschweig*, 1865.
[3] ARNDT, *Archiv f. mik. Anat.*, Bd. iii.
[4] SCHULTZE, *Archiv f. Anat. und Entwick. Gesch.*, 1878. *Archiv. f. mik. Anat.*, Bd. xvi.
[5] KÖLLIKER, *Handbuch der Gewebelehre*, Aufl. v., 1893.
[6] RETZIUS, *Studium in der Anatomie des Nervensystems*, &c., Stockholm, 1876.
[7] MEYNERT, *Strickers Handbuch*, 1870.
[8] BESSER, *Archiv f. path. Anat. und Physiol. u. Klin. Medic.*, Bd. 36, 1866.
[9] GERLACH, *Strickers Handbuch*, Leipzig, 1871.
[10] GOLGI, Numerous well-known contributions; collective papers under the title, *Untersuch. über d. fein. Bau d. Central. u. peripher. Nervensystems*, Jena, 1894.
[11] FOREL, *Archiv f. Psychiat. u. Nervenkrankheiten*, 1887.
[12] RETZIUS, *Biologische Untersuchungen*, Neue Folge, 7 Bde., 1890-95.
[13] HIS, *Archiv f. Anat. u. Phys.—Anat. Abth.*, 1890.
[14] CAJAL, Numerous contributions since 1893.
[15] KÖLLIKER, *Handbuch der Gewebelehre*, Aufl. vi., 1896.
[16] v. GEHUCHTEN, *Système Nerveux de l'homme*, 1893.
[17] LENHOSSÉK, *Der feinere Bau des Nervensystems*, Aufl. ii., 1895.
[18] EHRLICH, *Deutsch. Med. Wochenschr.*, No. 4, 1886, p. 49.
[19] ARONSOHN, *Beiträge z. Kenntniss d. Central. u. periph. Nervenendig.* Inaug. Diss., Berlin, 1886.
[20] BETHE, *Arch. f. mik. Anat.*, Bd. 46, 1895.

PLATE I.

FIG. 1.

FIG. 2.



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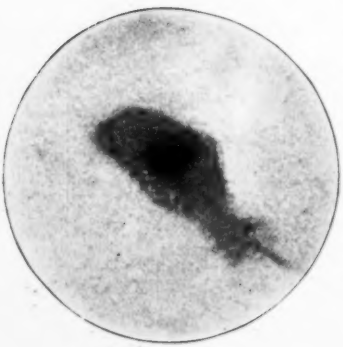

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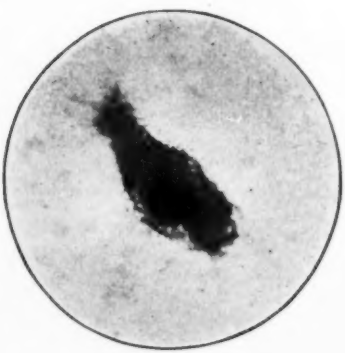

FIG. 5.

PLATE II.

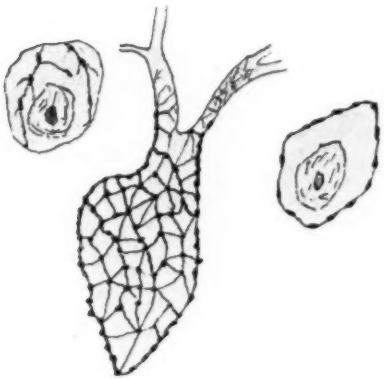

FIG 1.

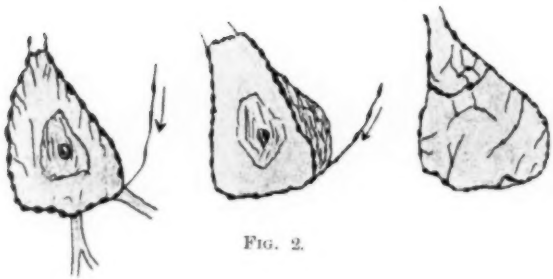

FIG. 2.

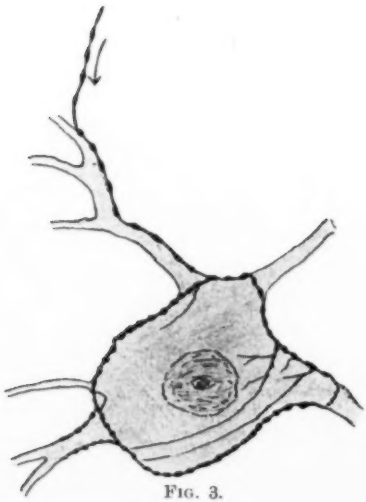

FIG. 3.

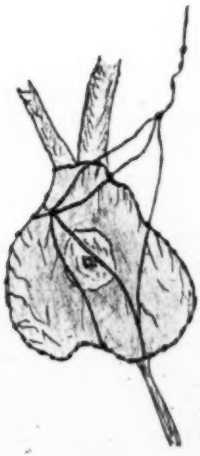

FIG. 4.

[21] Meyer, (1) *Arch. f. mik. Anat.*, Bd. 46, 1895; (2) *Arch. f. mik. Anat.*, Bd. 47, 1896.
[22] Meyer, *Über d. Function d. Protoplasm-Fortsätze d. Nervenzellen.—Abhandl. d. Sächs, Ges. d. Wiss*, 1898.
[23] Auerbach, *Neurol. Centralbl.*, 1898.
[24] Bethe, *Pflügers Archiv*, Bd. 68, 1897; *Morph. Arbeiten von Schwalbe*, Bd. 8, Hft. 1; *Archiv f. mikr. Anat.*, Bd. 44, 1897; *Archiv f. mikr. Anat.*, Bd. 50, 1 & 2 Mitteil.; *Archiv f. mikr. Anat.*, Bd. 51, 3 Mitteilung.
[25] Golgi, *Intorno alla struttura delle cellule nervose*, Paris, 1898.
[26] Held, *Beiträge z. Struct. d. Nervenzellen u. ihrer Fortsäze*, I. Mitteilung, *Arch. f. Anat. u. Phys.—Anat. Abth.*, 1895; II. Mitteilung, *ibid.*, 1897; III. Mitteilung, *ibid.*, 1897, suppl.
[27] Hill, "*The Chrome-Silver Method*," Brain, vol. xix, 1896.
[28] Held, "*Die centr. Bahnen d. N. Acustic, bei der Katze*," *Archiv f. Anat. u. Phys.—Anat. Abth.*, 1891; "*Die centr. Gehörleitung*," *Arch. f. Anat. u. Phys.—Anat. Abth.*, 1893.
[29] Kölliker, *Handbuch der Gewebelehre*, Aufl. vi., Bd. 2, 1896.
[30] Cajal, *Beitrag z. Studium d. Med. Oblongata*, 1896.
[31] Meyer, *Über d. Funct. d. Protopl. Fortsätze*, 1898.

EXPLANATION OF THE PLATES.

We are indebted to Mr. J. E. Barnard for the photo-micrographs, which have been taken from our original preparations.

Plate I.

Photomicrographs, illustrating the form of nerve termination described in the text.

Figs. 1 and 2.—Swift ⅛ dry objective. No. 2 ocular. Camera extension, 7 feet. 750 diameters. A nerve-ending from the cerebral cortex of a guinea pig; fig. 1 shows the network in focus; fig. 2, the peripheral zone in focus. In this termination there is no obvious evidence of a cell.

Fig. 3.—Swift ¼ dry objective. No. 2 ocular. Camera extension, 7 feet. 350 diameters. A nerve-ending from the nucleus trapezoides of a rabbit. Here the cellulipetal fibre is seen breaking up into a basket arrangement and enclosing a cell.

Figs. 4 and 5.—Same magnification as figs. 1 and 2. A cell from the nucleus of the posterior quadrigeminal body of a rabbit, enclosed in the terminal net-work. These figures show the appearances presented on altering the focus, so as to reveal *(a)* the peripheral zone, *(b)* the enshrouding network.

Plate II.

Diagrammatic representations of the termination and cells.

Fig. 1.—Cell from posterior quadrigeminal body.
Fig. 2.—Cells from cerebral cortex.
Fig. 3.—Cell from optic thalamus.
Fig. 4.—Cell from cerebral cortex.

A CONTRIBUTION TO THE LOCALISATION OF MUSCLES IN THE SPINAL CORD.

BY H. CAMPBELL THOMSON, M.D.LOND., M.R.C.P.

Physician to Out-Patients to the Hospital for Epilepsy and Paralysis, Regent's Park; Pathologist to the Middlesex Hospital.

DURING the past three years I have had numerous opportunities of examining cases of injuries to the spinal cord, some of which have been particularly instructive with regard to the relative localisation in the grey matter of muscles of the upper extremity.

The cases which I have found most valuable for this purpose are those in which the chief injury has apparently been due to hæmorrhage into the grey matter, for in many of these the lesion is sharply localised, and by careful comparison of the muscles which are affected in different cases, some conclusions may be arrived at regarding the different levels from which the muscles are supplied, and a finer analysis is thus more possible than in cases where the injuries are more diffuse.

An interesting case of this description was admitted into the Middlesex Hospital in 1897, under the care of Mr. Henry Morris, who kindly allowed me to make use of the notes.

The patient was a man who had injured his spine by diving into a swimming bath and striking his head against the bottom.

On admission there was weakness of all four limbs, which was rather more marked on the right side than on

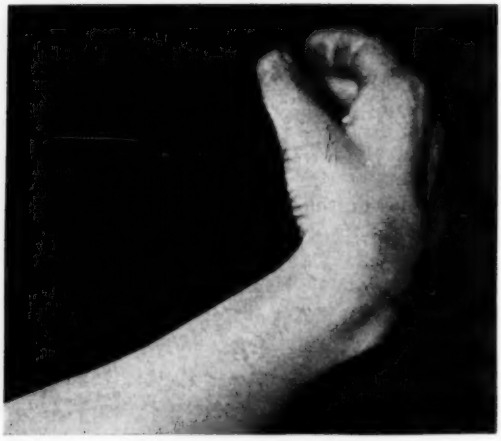

FIG. 1.
Showing power of flexing the wrist.

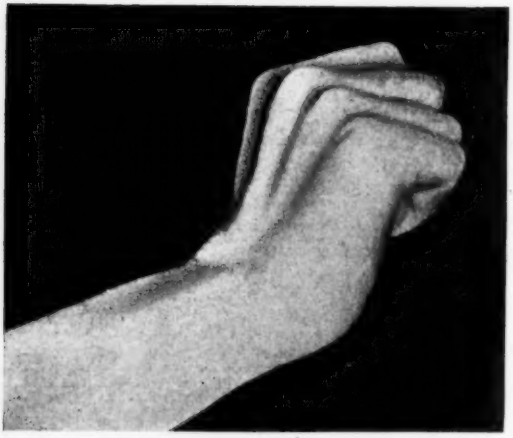

FIG. 2.
Showing over extension of wrist when attempting to flex the fingers.

the left, and it was also noted that the right hand seemed quite helpless.

Within a day or two the general weakness of the limbs almost entirely passed off, but the paralysis of the hand remained.

On examination the movements of the wrist joint were found to be normal (figs. 1 and 2), but there was complete loss of power in all the muscles attached below this level, *i.e.*, the long flexors and extensors of the fingers and thumb; the interossei and the small muscles of the thumb. No movement whatever could be made with these muscles, and whenever flexion of the fingers was attempted, a very strong over-action of the extensors of the wrist took place (fig. 2).

Rapid wasting also occurred in the paralysed muscles.

In a short time, however, some improvement began to take place, especially with regard to the long flexors of the fingers and the small muscles of the thumb, and both these groups acted to a moderate faradic current, but the power of flexing the first finger remained less than that of the other three.

The long extensors of the fingers and thumb and the interossei did not, unfortunately, join in this recovery, but gradually wasted and lost their irritability to the faradic current.

After the patient was able to leave the hospital he attended regularly for some weeks to receive electrical treatment, but finally, at his own request, this was discontinued.

At the time he ceased attending, it was evident that no further recovery was very likely to take place, and the condition was as follows:—

The long extensors of fingers and thumb The interossei	... Remained paralysed.
The long flexors of fingers and thumb	... Partially recovered.
The small muscles of the thumb	... Recovered.

With regard to the nature of the lesion in this case, there was no doubt a slight transverse injury to the cord at the time of the accident, as shown by the temporary paralysis of all four limbs, but the more permanent effects were in all probability caused by a localised hæmorrhage into the grey matter which evidently chiefly affected those nuclei which govern the muscles of the hand, and caused no disturbance of sensation beyond some slight thermal anæsthesia, such as so often accompanies a lesion of this description. The interest of the case, with regard to the localisation of muscles, lies in the fact that while the muscles of the wrist entirely escaped, all those below it were more or less affected, and we therefore see that the nuclei which supply the long flexors and extensors of the hand are definitely separated from, and probably situated at a lower level than, those which supply the muscles of the wrist-joint.

A comparison of this case with another one of a somewhat similar nature, which, through the kindness of Mr. Andrew Clark, I have recently had an opportunity of examining, further shows the relative position which the nuclei of the long extensors and flexors occupy in the spinal cord.

The case was one in which there was wasting of the muscles of both hands, accompanied by extensive loss of thermal and painful sensations, while those of touch were preserved, and therefore, like the first case, the lesion was probably one of hæmorrhage into the grey matter. But, whereas in the first case the long flexors and extensors of the fingers were affected with the small muscles of the hand, while the muscles of the wrist escaped; in the second case the small muscles of the hand were alone affected, and the long extensors and flexors escaped completely.

The fact that in the one case the flexors and extensors of the fingers shared the same fate as the small hand muscles, and in another shared the fate of the wrist muscles, clearly points to the supposition that their nuclei are placed in an intermediate position between those of the other two.

The result of the first case also suggests that there

is probably a close relation between the long extensors and the interossei, since these were the two groups which remained permanently paralysed.

Another case of injury to the spinal cord, the notes of which I have unfortunately mislaid, led me to think that the motor cells for the extensor carpi radialis are situated at a higher level than those of the extensor carpi ulnaris.

The following diagram illustrates the relative positions of the various nuclei as suggested by the cases which I have observed.

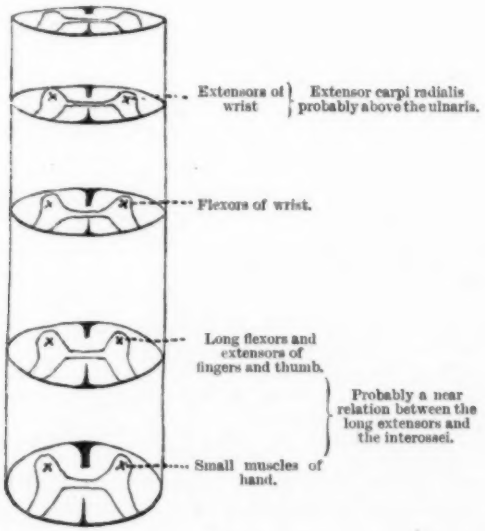
Extensors of wrist
Extensor carpi radialis probably above the ulnaris.
Flexors of wrist.
Long flexors and extensors of fingers and thumb.
Probably a near relation between the long extensors and the interossei.
Small muscles of hand.

FIG. 3.

It is generally assumed that the group of muscles supplied by a given root is the same as that supplied by the corresponding segment of the spinal cord; but this is not necessarily so, and the above cases indicate that the representation of muscles in the cord is much more widely distributed than that of the roots, and that the latter are made up of fibres converging from their anterior cornua at different levels of the cord.

With regard to the root distribution, according to most clinical records the extensors of the wrist and fingers are supplied from the seventh cervical root, the flexors of the wrist and fingers from the eighth, and the interossei and other small muscles of the hand from the first dorsal, whereas the cases above quoted show that in lesions of the anterior cornua the flexors and extensors of the fingers may be affected independently of the muscles of the wrist, and also independently of the small muscles of the hand.

HYPNOTISM: A REPLY TO RECENT CRITICISMS.

BY J. MILNE BRAMWELL, M.B.

At the present day, although the reality of hypnotic phenomena is generally conceded, wide differences of opinion exist as to the nature of hypnotism, and its value and safety as a therapeutic agent. Thus, in "Hypnotism in Court" and "Hypnotism," two articles which appeared in the July and October numbers of the *Journal of Mental Science* for 1898, the views expressed are not held by the majority of those who practise hypnotism. As the articles referred to, both apparently from the same pen, are unsigned, I will, for convenience' sake, call the writer X.

First, I wish to draw attention to a personal point; X, in referring to my address to the Psychological Section of the British Medical Association, says: "The bold attempt of Dr. Milne Bramwell to prove there are no drawbacks to the therapeutic use of hypnotism is, however, a challenge which should be promptly met." Now I have never, either in writing or speaking, asserted that there were no drawbacks to the therapeutic use of hypnotism, but, on the contrary, have constantly pointed out that many existed. For example, in this country hypnotism is rarely resorted to until other remedial measures have failed, and this greatly decreases its chance of success. Further, I have never denied that hypnotism, through ignorance or malice on the part of the operator, might be so misused as to do harm; this risk, however, is not only grossly exaggerated, but falls far short of that associated with ordinary medical practice.

What I really said was totally different, namely, that, as far as my experience went, the employment of hypnotism by medical men acquainted with the subject was devoid of danger. In support of this I cited Forel's assertion that he, as well as Liébeault, Bernheim, Wetterstrand, van Eden, de Jong, Moll, and the other followers of the Nancy school, had never seen a single instance in which mental or physical harm had been caused by hypnosis. No complete record of their cases has been published, but the number certainly exceeds fifty thousand. On October 15, 1898, Forel informed me that he still held this opinion, and had never observed even the slightest inconvenience from hypnotic practice. Further, I have watched the work of

nearly all of those cited by Forel and have seen nothing opposed to his statement.

I could add many names to Forel's list, but will content myself by quoting one or two in this country, choosing them for their connection with asylum practice.

In 1890, Dr. Percy Smith and the late Dr. A. T. Myers published an account of the hypnotic treatment of twenty-one insane patients in Bethlem Hospital, I am authorised by the former to state that no harm was done.

Dr. Woods, of Hoxton House Asylum, has hypnotised over a thousand persons, many of whom were insane. He recently informed me "that he had never in his own practice seen any evil effects, either mental or physical, caused by the use of hypnotism."

I have also Dr. Outterson Wood's permission to state that, while he acted as Honorary Secretary to the British Medical Association Committee for the Investigation of Hypnotism, he performed several hundred experiments and arrived at the following conclusions with regard to hypnotism:—(1) Its phenomena were genuine; (2) It was of distinct therapeutic value; (3) Its use in skilled hands was absolutely devoid of danger.

My position might be attacked in two ways: (1) By attempting to prove mal-observation; (2) The facts being granted, by showing that they did not warrant the conclusions drawn from them. These, the only two points at issue, X entirely ignores. He says: "Very many observers have seen cases in which hypnotism has been followed by very definite and distinctly evil results. Many instances of this kind have been recorded, and good service would be done by their collection and tabulation, as a check to future assertions of this kind." Granting that this be true, what connection has it with the point in dispute? If a group of surgeons performed a certain operation fifty thousand times without a death, would they not be justified in inferring that in their hands, at all events, it was devoid of danger? Would their position be weakened by the "collection and tabulation" of the failures of others?

There remain for discussion the hypnotic dangers referred to by X, namely (1) evil results recorded by other observers, and (2) those occurring in his own practice.

(1) Harm reported by others.

As regards this group, X does not cite a single authority or case; surely the "collection and tabulation" to which he refers should have been done beforehand, and the results presented in such a way as to enable one to judge of their value as evidence.

I have investigated every case brought to my notice where hypnotism was stated to have done harm, and in none has the

charge been proven. The two following may serve as a commencement of the collection X is desirous of making :—

(*A*) A patient, suffering from torticollis, who had been under my care for a few weeks, shortly afterwards underwent a surgical operation; an important blood-vessel was accidentally cut and about ten days later she died from secondary hæmorrhage. I was informed that the surgeon asserted the hæmorrhage and death were due to the fact that the patient's constitution had been weakened by hypnotism. If this were true, the relatives felt that they had contributed to the death by permitting the patient to try hypnotic treatment, and I was asked whether I could in any way reassure them. This I had little difficulty in doing. I informed them (*a*) that hypnotism did not weaken the constitution; under its influence Esdaile had reduced the mortality in the removal of the tumours of elephantiasis from 50 to 5 per cent., and my own operative cases had all done well; (*b*) even granting that hypnotism could weaken the constitution, it was unreasonable to suppose that it had spread from the patient to the surgeon and thereby caused him to accidentally cut a blood-vessel; (*c*) I mentioned the not unimportant fact that I had absolutely failed to hypnotise the patient.

(*B*) In April, 1897, a medical acquaintance told me I must alter my views as to the dangers of hypnotism, because, by its means, M., a Swiss medical man, had seduced eleven young female patients and had been condemned to five years' imprisonment. One of the victims, I was told, had committed suicide, and before doing so, had left a written statement of the fact that she had been seduced through hypnotism. Further, I was informed that these statements were confirmed by ample official evidence. This I asked to see, and received what was described as "the official testimony from Dr. Calonder, of the 'Advokatur-bureau,' in Chur, where the case was tried, stating that hypnotism had been abused in this shocking betrayal." My colleague continued, "I could also send you the four German newspapers sent me; but as they contain no details which I have not given you, you probably would not care to have them."

The following is a translation of the document in question :—

"In reply to your favour of the 26th inst., I hasten to inform you that, at the trial of Dr. M., no support was found for the assertion that he had employed hypnotism in the treatment of his patients.—(*Signed*) Dr. Calonder."

I asked for the German newspapers, and found that in the account of the trial hypnotism was not even mentioned. I then laid the matter before Professor Forel, of Zurich, who replied as follows: "Dr. M. has never hypnotised a patient. The question was not raised at the trial, and no one suggested that he had employed hypnotism. The entire history is an English invention. In Zurich no one has spoken or written of hypnotism in connection with Dr. M. He is a throat specialist—an ordinary erotic pig, and has long been known as such. I have written to

the Judge of the Supreme Court, who tried Dr. M., and he has sent me a written statement which confirms mine; this I now enclose, so that you may have a trustworthy document." Prof. Forel's letter and that of the Judge are still in my possession. They describe Dr. M.'s methods of seduction, but as these have nothing to do with hypnotism, and are not particularly fit for publication, I refrain from quoting them.

(2) X's Cases.

X says, in his special department, presumably asylum practice, the evils consequent on hypnotic influence are grave, and that the few cases in which it seemed desirable to induce hypnosis, were, in the end, apparently deteriorated in mental condition, and that the conservation of mental power, so urgently indicated was, in fact, endangered. From this he concludes that hypnosis involves a weakening of the power of self-control, and "is indeed, a shunting on one of those side tracks of disordered mental function of which insanity is the terminus." Here I may point out (1) that no figures are given, and the word "few" is an indefinite one; (2) the disease from which the patients suffered is not stated, nor the absolutely essential information given whether it was one likely to end in mental deterioration apart from the treatment; (3) X does not assert that mental deterioration resulted from hypnosis, but only that it apparently did so; (4) although X practically states that hypnosis leads to insanity, he does not tell us that insanity was actually produced.

It is to be regretted that X gives no detailed account of his cases, as thus, the dangers to which he refers are based more upon assertion than upon evidence which might be examined and judged. Granting, however, that mental deterioration actually resulted from X's hypnotic practice, one cannot help feeling that his personal and limited experiments do not warrant the conclusions he draws from them. Fortunately, it is not a recognised principle in medicine that those who fail have the right to cry "Halt!" to the successful.

According to X, hypnotism is related to hysteria, stupor, latah, &c.

(I.) *Hysteria.*—The supposed connection between hypnotism and hysteria, disputed by Esdaile and Braid more than fifty years ago, was restated by the Salpêtrière school, and successfully disproved by that of Nancy. X's attempt to revive an ancient fallacy is not likely to be successful, especially as he does not support it by a single observed fact.

At the International Congress of Experimental Psychology, London, 1892, it was generally recognised that susceptibility to hypnosis did not indicate hysteria or any other morbid condition in the subject. Hypnotism was found to increase the patient's volition; thus the cure was due to the intensification of an entirely normal moral process, and was not really dependent upon assist-

ance from without. It is true, as Myers has pointed out, that a superficial resemblance exists between the phenomena of hysteria and those of hypnotism. In hysteria certain nervous, vasomotor and circulatory changes arise, apparently as the result of irrational and unconscious self-suggestion in regions beyond the power of the waking will. The hypnotic self also can influence the nervous, vasomotor and circulatory systems to an extent unparalleled in waking life. Here, however, the phenomena are under the subject's own control, and he can be taught to evoke and terminate them at will.

According to X, self-control is weakened in hypnosis. This view is an old mesmeric fallacy which was successfully exploded by Braid fifty years ago. My patients, no matter how deeply hypnotised, can always choose between suggestions and reject what is distasteful. The supposed loss of self-control is assumed by the operator from the observation of laboratory crimes. Questioning in hypnosis, however, reveals the fact that there has been neither loss of consciousness nor of volition. This view I have put before the profession from time to time, and most of the experiments on which it is founded have been carefully checked by others. Some of these, carried out in conjunction with Dr. Hyslop, of Bethlem, are recorded in his work entitled "Mental Physiology."

X asserts that therapeusis admits the principle that lower tissue may be sacrificed to save a higher, but that it is distinctly bad practice to harm the higher organ to release the lower, as hypnotism does. This concerns ordinary rather than hypnotic practice. Most of the diseases which are treated hypnotically are central in their origin, as, for example, dipsomania, hysteria, obsessions, &c. In such cases it is the relief or cure of the higher organ which is at stake, and no sacrifice of any kind, either higher or lower, is involved. Lost or weakened self-control is at the root of the diseases just referred to, and, when hypnotism cures them, it does so by developing the patient's own volition.

If, however, we judge ordinary practice by X's standards, the administration of narcotics and anæsthetics must be condemned, and their employment for the relief of pain considered bad practice. X's opinion, I think, is not now generally held by the profession, although, in 1842, Dr. Copland asserted, at the Royal Medical and Chirurgical Society, that "pain was a wise provision of nature, and patients ought to suffer pain while their surgeons were operating; they were all the better for it, and recovered better."

(II.) *Stupor.*—Stupor has been divided into two forms—(1) Anergic and (2) Delusional.

(1) Anergic.—In typical cases there is blueness and swelling of the hands and feet, slow and feeble circulation, vacant expression, retention (in some cases incontinence) of urine and fæces, complete absence of mental function in the region of will, perception, memory, and often even of consciousness.

(2) Delusional.—This form is the outcome of profound melancholia, in which the mental activity is terminated by a melancholic delusion.

Stupor is supposed to be due to pathological changes in the brain and its blood-vessels; it is generally associated with grave disease and frequently has a fatal termination ("Dictionary of Psychological Medicine," Hack Tuke).

Again X contents himself with simple assertion, and omits to adduce a single fact in support of the supposed connection between stupor and hypnosis. Hypnotised persons do not turn blue, nor do their hands and feet swell; they do not lose control over the bladder or bowels. Their volition is unimpaired, their perception and memory are often abnormally acute and consciousness is rarely, if ever, lost. Hypnosis is not associated with profound melancholia, nor does it end in a cessation of mental activity. On the contrary, the hypnotised person may be able to solve mental problems of great difficulty which are beyond the range of his normal powers. Again stupor, unlike hypnosis, cannot be originated, guided or terminated at will. Further, hypnosis does not arise from pathological changes in the brain and its blood vessels, nor has it a fatal termination.

(III.) *Latah.*—X talks of latah as if it were a disease, but, as Moll has pointed out, the word latah designates the sufferers from the complaint, not the disease itself. Latah, according to Gilles de la Tourette, is a "tic" characterised by certain abnormalities of movement, particularly the imitation of gestures, and by certain anomalies of speech, such as the use of bad language, and the repetition of the last words uttered in the patient's presence. Bamberger states that violent contractions of the flexors and extensors of the muscles of the leg take place when the patient attempts to stand, and thus a jumping movement is produced.

According to Clifford and Talcott Williams, the condition is latent in the Malay race and can be induced by startling them and making them jump. They will then imitate actions which are painful, dangerous, or obscene. The Malays are a race particular to a fault about all matters of personal modesty, but a latah Malay woman will strip naked in a public place at the casual invitation of a passing stranger.

In attempting to establish an analogy between hypnotism and latah, X, as usual, states an old fallacy without adducing a single fact in support of it. His theory is apparently a reproduction of that of Dr. Gillmore Ellis (*The Journal of Mental Science*, January, 1897, p. 32). Neither writer seems to be aware of the fact that similar views were expressed at an earlier date by several Continental observers, and their unsoundness demonstrated by Moll and others. The fallacy appears to have arisen from untrained and superficial observation. Thus, it has been noticed that both the latah Malay and the hypnotised person imitate gestures; the conditions, therefore, are assumed to be similar.

The circumstances, however, under which they do so are widely different. The moment a "latah" is startled he will at once imitate any gesture made before him by anyone. He will also mimic the swaying motion of wind-shaken boughs as readily as the actions of a human being (Clifford). No verbal suggestion is necessary. On the other hand, hypnotised subjects never copy movements made before them unless they have been trained to do so, and then only those of the hypnotiser. Again, hypnotised persons, even despite energetic suggestion, will not imitate actions which involve real danger, actual indelicacy, or obscenity, nor do they make use of bad language. Further, the hypnotic subject, unlike the latah, does not hop about like a kangaroo the moment you startle him. Moreover, hypnosis can be terminated at will, but the disease from which the latah suffers is uncontrollable and frequently incurable.

Other writers besides X have attempted to establish a connection between hypnosis and many and widely differing diseased conditions. In every instance the fallacy has been due to the imperfect observation of some isolated hypnotic phenomenon. Benedikt's theory is fairly characteristic of this class. In his view of hypnosis, its numerous, varied and complex mental and physical conditions are ignored, and it is simply classified as an artificially induced catalepsy, because in hypnosis the muscles can be rendered rigid by suggestion.

Such theories have the merit of simplicity, and possess the additional advantage that they can be made without careful observation or prolonged study. What should we think, however, of the observer who described normal human life as simply an artificially induced sneeze, because the act of sneezing could be readily invoked by the presentation of a pinch of snuff!

Moll draws attention to the fact that hypnosis has been compared to the most dissimilar forms of mental disease. Those who do so, he says, forget amongst other important things that susceptibility to suggestion is the chief phenomenon of hypnosis, and that this is far from being an essential characteristic of mental disorder. We can produce paralysis, stammering, or pain by suggestion, but hypnosis itself is none of these things. Moll points out that it is illogical to call hypnosis a disease because morbid imitations of it are found. Yawning itself is not a disease, although attacks of it occur, which are morbid in character. Thus, we are no more justified in inferring that the hypnotised subject suffers from hysteria because he can imitate its symptoms than in concluding that an actor is mad when he portrays insanity. Further, hypnosis is entirely under the control of the operator, or of the subject himself, but there is no disease which can be originated, guided and terminated at a moment's notice.

Faith-healing.—X asserts that "therapeutically hypnotism is related to various forms of faith-healing, the limitations of which are fairly well-known and recognised." X does not define faith-healing, and it may be useful to supply his omission.

According to the Bible, the power of healing is one of the attributes of the Deity which is at the service of those who solicit it, and believe their prayer will be granted. Here two factors are involved, the power to cure possessed by the operator and faith on the part of the patient. The former may be eliminated from present day faith-healing, which is essentially characterised by a belief in what does not exist, *i.e.*, the curative power of the operator. X's theory, that hypnotic phenomena are due to emotional states associated with faith, was held by Braid in the earlier part of his career, and is still regarded as correct by certain later observers. Recent investigations, however, have done much to discredit this view, and a short account of its origin, together with the changes which have since taken place in hypnotic theory may not be without interest.

In Braid's day, so-called mesmeric phenomena were supposed to be due to external agencies, *i.e.*, a mysterious force existing in the operator, and in various metals, magnets, &c. Braid showed how identical phenomena could be induced in the absence of the supposed exciting causes, if the patients were led to believe that these were present. For example, the cures attributed by Dr. Perkins to the action of his "metallic tractors" were equally successfully obtained by Dr. Haygarth's pieces of wood. Braid concluded that in all these things faith, expectation and concentrated attention were the only curative agents. Further, he thought, the action of medicines might partly be explained in the same way, and that the mental element associated with their administration had been far too much ignored. All medicines had a two-fold action; one due to their physical properties, the other to the patient's expectation of a definite result. This mental impression accounted for the varying estimates as to the value of certain drugs. Thus, at one time a universal favourite was supposed to cure everything, then was regarded as valueless, only later to regain its former position. The drug itself did not change, only the belief or confidence of those who prescribed, and those who took, it.

Elliotson also pointed out that, owing to the mistaken way in which medicines were frequently administered, their curative effects, if any, must be due to the faith of the patient. Some physicians, he said, had actually fixed doses for each medicine, one, for example, never gave more than two grains of iodide of potash, even if it produced no effect. Others changed their prescriptions every few days and the patients thought they had tried everything, whereas they had in reality tried nothing. Thus, the drugs had not the slightest chance of benefiting the patient, as they were neither given in sufficient doses nor continued long enough.

Braid's work being practically forgotten, certain members of the Salpêtrière school revived many of the old errors of the mesmerists, and magnets, metals and medicines in sealed tubes,

were once more supposed to be endowed with marvellous properties. These fallacies were for the second time exposed by the members of the Nancy School. They, like Braid in his earlier days, attributed the phenomena to emotional conditions excited in the brain of the patient. Everything was due to what they termed "suggestion." The phrase soon became a fashionable one, and was considered to explain not only the curative powers of hypnotism, but also many therapeutic results which were usually attributed to the action of drugs.

Thus, Hack Tuke, in speaking of Sir Andrew Clarke, said: "His favourite drugs were bi-carbonate of potash and a vegetable bitter, but neither drugs nor diet formed the central factor of his treatment, or explained its success. 'Suggestion' lay at the root of it all. The term, however, is too mild unless understood in the technical sense in which it has been employed in recent times. In short, Sir Andrew out-Bernheimed Bernheim; he was, in a word, the most successful hypnotist of his day."

According to Sir Samuel Wilks: "To sit down in one's chair daily and write on a piece of paper the name of some drug for every ailment without exception, which comes under our observation is, in the present state of medicine, an absurdity, and is simply a pandering to human weakness. I do not say that drugs are not useful in a moral sense, I am merely contending that the method is not scientific, as we usually apply this term. I know of no more successful practitioner than the late Sir William Gull, and his treatment was rational, but he did not credit any particular drug with the properties ascribed to it by the patient. His prescriptions very often consisted of nothing but coloured water." Sir Samuel describes a "sixpenny doctor" at a cheap dispensary, who saw on an average seventy patients each evening, and whose almost universal medicine was a mixture composed of sulphate of magnesia, burnt sugar, and infusion of quassia. The following were his directions for successful practice: "Always give medicines which produce appreciable effects; then, also, the mixture must taste like medicine, and if it have a bad smell the patient will be better satisfied."

Sir Samuel states that "changes in the pathological views of disease have caused the whole method of its treatment to be altered again and again, and that further chemical knowledge frequently shows that the drugs we employ do not possess the qualities that we have been in the habit of attributing to them."

Putting aside for the moment the question of the connection between hypnotism and faith-healing, we find Hack Tuke and Sir Samuel Wilks practically reproducing the views of Braid and Elliotson to the effect that in many instances drugs owe their supposed therapeutic value to the faith of the patient. In typical cases the supposed curative agent is stated to be either non-existent or inert, as, for example, in the "coloured water" of Sir William Gull and the magnets of Benedikt. The latter still holds that magnets possess extraordinary therapeutic powers, despite

the fact that scientific investigation has shown them to be devoid of the properties he attributes to them, and that they would be equally powerful if composed of pieces of wood. His method of treatment is, in fact, a reproduction of Perkins' tractors already referred to.

I do not believe that the greater part of medical treatment merits Sir Samuel Wilks' description, but certainly some of it does. It is thus identical with present day faith-healing, as the curative effects are due to the patient's belief in what does not exist. Such methods of treatment have their practical drawbacks, as advancing knowledge is ever likely to rob them of their power. Harness' electropathic belts undoubtedly sometimes caused the disappearance of functional nervous disturbances; but, to my personal knowledge, these in some cases immediately reappeared after the Harness' exposure. Would Benedikt's magnets be still efficacious if all his patients knew they were really powerless? Would Sir William Gull have continued to be equally successful had Sir Samuel Wilks' description of his method of treatment been universally accepted? Would it not be better either to substitute "suggestion" alone for "coloured water" and magnets, or to employ instead of them remedial agents of undoubted value?

In order to bring hypnotism under the head of faith-healing, one must be able to show that the patient believes in the operator's power of healing, and that nothing beyond faith is involved. Since the days of Braid, both physician and patient have learnt that the curative power is due to changes in the patient's own nervous system, and is independent of any mysterious power possessed by the operator. Faith is not necessary for the induction of hypnosis. Some who believe they can be easily hypnotised prove refractory, while others who are intensely sceptical are influenced at the first attempt. Further, in self-hypnosis, the influence of the operator is entirely eliminated, and if cure be due to faith, it is to the patient's faith in himself. Again, hypnosis is not an imaginary condition; even X not only admits its existence, but also warns us of its dangers.

Later, Braid recognised the complex and varied nature of hypnotic phenomena, and that these could not be explained by faith, suggestion, or concentrated attention alone. Many later observers have arrived at identical conclusions. It is now recognised that in hypnosis the attention may be directed to many points at once, and that suggestion is simply the artifice by means of which the phenomena are evoked. Wide differences exist between the results of suggestion in emotional states and suggestion in hypnosis. Thus:—

(A) *Suggestion in Hypnosis.*

(1) Once hypnosis has been induced, a wide range of phenomena, both mental and physical, can be evoked at any time, and—with the consent of the subject—by anyone. A considerable number of phenomena can be simultaneously evoked in the same subject.

(2) One phenomenon can be immediately changed into its opposite, *e.g.*, muscular rigidity into paralysis, anæsthesia into hyperæsthesia, &c.

(3) Hypnotic phenomena can be terminated at will.

(4) The date of the appearance of a given phenomenon can be delayed, *i.e.*, it can be suggested during hypnosis that the phenomenon shall not appear till twelve months afterwards.

(5) The suggestion will invariably be responded to, subject to the two important limitations that it contains nothing in opposition to the patient's moral sense, and that it is not beyond the range of his hypnotic powers.

(6) Under the conditions just mentioned, the exact nature of the response can be predicted, *i.e.*, similar stimuli produce identical results.

(7) Patients, who readily respond to suggestions when hypnotised, are frequently the very subjects who have for years resisted suggestion in the normal condition, even when this has been associated with emotional states. For example, a patient who had long suffered from dipsomania received many and varied suggestions in the normal state. All these, however, produced no result; yet, after a few weeks' hypnotic treatment, the patient abandoned the alcoholic habit, and, although over ten years have passed since then, has not relapsed.

(8) Hypnotic suggestion tends to gain strength by repetition.

(B) *Suggestion by Means of Faith and other Emotional States.*

(1) The resultant phenomena are usually isolated ones, or, at all events, much more limited in number than those which can be simultaneously evoked in hypnotised subjects. Further, they cannot be produced by anyone. Thus, the subject who has been influenced by the touch of a king would probably be unresponsive to that of a peasant.

(2) One phenomenon cannot be immediately changed into its opposite, without an alteration in the emotional state which had produced it.

(3) Emotional phenomena cannot be terminated at will.

(4) The date of the appearance of the phenomena can rarely be delayed and fixed.

(5) The phenomena are evoked with less certainty than in hypnosis; an emotional state which will produce a physical effect in one subject will produce nothing in another.

(6) Identical emotional states do not always produce similar physical phenomena. On the contrary, opposite conditions are frequently evoked in different subjects by identical emotions, *e.g.*, fear will paralyse one, and excite violent muscular contractions in another.

(7) Patients who are unaffected by curative suggestions in the normal state readily respond to them when hypnotised.

(8) An emotional suggestion frequently loses strength by repetition, *i.e.*, a subject may readily come to disregard former fears.

While X's fallacies were long ago exploded by the Nancy school, its views, in their turn, are now being attacked. Concentration of attention and suggestion are no longer generally regarded as explanatory of the hypnotic state. Personally, I am acquainted with no theory which satisfactorily explains hypnotism as a whole. The subliminal consciousness theory of Myers certainly marks an important advance, and gives one a clearer conception of the mental and physical states involved in hypnotic phenomena. It does not satisfactorily explain, however, whence the hypnotic subject derives his powers, nor how suggestion evokes them. X's treatment of Myers' theory is scarcely a scientific one. In reference to hypnotism, X says: "Hypotheses as to its nature may be useful, but should be carefully based on known facts, and not complicated by dialectical subtleties, such as the 'subliminal consciousness.'" Here, it is to be noted, that while X does not adduce a single fact in support of his widely varying hypotheses as to the nature of hypnotism, he is ignorant of, or ignores, those on which the subliminal consciousness theory is based. The theory is not Myers' alone, though his is probably the clearest statement of it. Many writers in different countries have recorded observations on which it is founded. X's assertion would have carried more weight if he had, at the same time, given us the result of his examination of the experiments of Azam, Delbœuf, Pierre Janet, Max Dessoir, William James and others. It is to be noted that William James states that the principle upon which the subliminal consciousness theory depends is now largely admitted by science.

According to X, the assertion that it (hypnotism) unlocks or redevelops some latent organic endowment is beyond possible proof, and is opposed to all evidence of organic evolution. That the hypnotised person possesses powers over his organism, which are not shared by the ordinary individual, instead of being beyond possible proof, is now one of the commonplace facts of science. The ordinary individual cannot induce anæsthesia at will, nor can he select painful sensations from amongst a group of others and prevent these alone from reaching consciousness. To the deeply-hypnotised subject, however, this feat is an easy one, as I have frequently demonstrated to members of our profession.

X says: "We recall a visit to the Zurich Asylum, where Professor Forel has long studied the phenomena of hypnotism, and where many of his 'subjects' could be seen amongst the members of the staff. Only one patient, an habitual drunkard, was then sensitive to the hypnotic influence exerted by Professor Forel." As X in the next sentence states that he personally had only employed hypnotism in a few cases and with unfortunate results, those unacquainted with Forel's work would naturally conclude that Forel too, like X, had found hypnotism of little

therapeutic value, and so abandoned it. This imputation is keenly resented by Forel. The following are extracts from two letters which he wrote to me from Chigny, in October and December, 1898. He said: "I have left Zurich as I found the work too much for me, but it is altogether false that I have given up hypnotism. On the contrary, I treat patients here by this method, and continue to get very good results. The *Journal of Mental Science* has stated a thing which is false. I have hypnotised over a thousand persons and have influenced about 95 per cent. The therapeutic results are excellent. The allusion in the *Journal of Mental Science* is a bad joke, and reminds me of the Englishman who in passing through France saw an ugly girl with red hair at a station and concluded that all French girls were ugly and had red hair. I have no recollection of the visit referred to, but if I then hypnotised an alcoholic that does not prove that I did not hypnotise 999 other persons. In any case, it is false to pretend that amongst the numerous patients I have hypnotised one alone was susceptible; probably this gentleman understands nothing about the subject."

Forel is the author of "Der Hypnotismus, seine psycho-physiologische, medicinische, strafrechtliche Bedeutung, und seine Handhabung," which has already reached its third edition. Until recently he was the editor of the *Zeitschrift für Hypnotismus* and has contributed many articles on hypnotism to that and other medical journals. It was, in fact, owing to his researches that the great hypnotic movement spread from France to Germany.

X states that many of Forel's "subjects" could be seen among the members of the staff, but does not mention that they were hypnotised for physiological and psychological experiment. Forel found, for example, that he could produce redness of the skin and urticaria by suggestion, and also action of the bowels. He records several instances in which he succeeded in causing the appearance or disappearance of the menses during hypnosis, and others in which the same results were brought about by post-hypnotic suggestion.

In the *Zeitschrift für Hypnotismus*, for 1898, Dr. Inhelder, formerly Assistant Physician to Professor Forel, records the experiments made with hypnotised warders, both male and female, for ten years (1887-1897), in the night watching of dangerous lunatics. Hypnotised warders were trained to sleep by the bedside of dangerous patients and to awake the instant the patients attempted to get out of bed. Sounds which had no import with reference to the warder's duties were inhibited by suggestion. Warders in this way sometimes performed night duty for six months, and worked hard all day without showing signs of fatigue. Dr. Inhelder gives a detailed account of these cases and states that the results were uniformly successful. No accident of any kind occurred.

In reference to these experiments, Forel claims that they

were entirely without risk, and were conducted in such a rigorously scientific manner as to exclude all possible sources of error.

When I visited Forel in 1892, he successfully repeated several of these experiments, and also showed me a number of patients who were undergoing hypnotic treatment.

The Therapeutic Value of Hypnotism.

In 1891, Forel published the following list of diseases, which, in his experience, responded best to hypnotic treatment: All forms of hysteria, including hystero-epilepsy, neurasthenia, nocturnal terrors of children, chorea, enuresis nocturnalis, stammering, insomnia, headache, neuralgia, sea-sickness, vomiting of pregnancy, psychical impotence, perverse sexual disturbances and bad habits of all kinds, alcoholism and morphinism, chlorosis, menstrual disorders, constipation, loss of appetite, nervous dyspepsia, chronic muscular and articular rheumatism.

This list, he said, was by no means complete, but would be sufficient for any fresh worker in the field of hypnotism to begin with.

Hysteria.—According to many authorities, hypnotic treatment gives the best results in—

(1) The convulsive attacks of *grande hystérie*, and in the symptoms which sometimes persist after these attacks (paralysis, contractures, spasms, anæsthesia, amoroses, &c.).

(2) Monosymptomatic hysteria (monoplegia, hysterical mutism, aphonia, hiccough, cough, blepharospasm, discromatopsy, &c.).

(3) The various manifestations of ordinary hysteria (insomnia, dyspepsia, visceral and menstrual troubles, neuralgia, &c.).

(4) Mental troubles of a hysterical nature (perversion of sentiment, obsessions, irresistible impulses, hallucinations, melancholia, maniacal excitement, &c.).

Bérillon states that for three years he watched the hypnotic treatment of *grande hystérie* by the late Dr. Dumontpallier at the Pitié Hospital. In the majority of cases, not only did the convulsive attacks disappear, but the other symptoms also. Some of these patients are now in excellent health, married and mothers of families, while others occupy important posts.

Dipsomania.—Florel claims to have obtained excellent and durable results in this disease, and there are few, if any, of those engaged in hypnotic work who are unable to report similar successes. Ladame draws special attention to three cases treated by Florel. All had had attacks of delirium tremens and were inmates of his asylum. They were extremely difficult to manage and expressed a determination to resume drinking as soon as they were liberated, but, despite this, complete recovery followed hypnotic treatment.

Vicious and degenerate children.—Bérillon and others assert that many carefully observed facts prove the therapeutic value of suggestion in the following diseases of children: Incontinence of urine and fæces, nervous "tics," nocturnal terrors, chorea, onanism, blepharospasm, &c.

Obsessions.—Hypnotic treatment has yielded brilliant results in various forms of obsession, and the following, amongst others, cite successful cases: Liébeault, Schrenck-Notzing, Hecker, Wetterstrand, de Jong, van Eeden, van Renterghen, Bernheim, Gorodichze, Voisin, Burot, Mavrokakis, Bourdon.

Neurasthenia.—Schrenck-Notzing records 228 cases treated by Brügelman, Bérillon, Bourdon, Voisin, Burckhardt, Forel, Ringier, Hecker, Ritzmann, Bourru and Burot, Stadelman, von Corval, Bernheim, van Renterghen, Wetterstrand, himself, and others. Of these, 31 per cent. were reported cured.

Menstrual disorders.—Successful cases of amenorrhœa, dysmenorrhœa, and menorrhagia are cited by Forel, Tyko Brunnberg, Delius, Wetterstrand, Voisin, Bérillon, Gascard, Journée, Maradon de Monthyel, Burot, Dècle, &c. Many of the cases were of long standing, and had resisted other methods of treatment, while their recovery is confirmed by later reports.

Insanity.—From 1880 till his death, in 1898, Voisin devoted himself largely to the treatment of mental disorders by hypnotic suggestion, and claimed to have cured many cases of undoubted insanity. The majority of the patients suffered from mania, or from melancholia with hallucinations, suicidal impulses, and delusions of persecution. Many of the cases are of extreme interest and carefully reported. The following is an example:—

T., aged 40. Suicidal melancholia with hallucinations. When the patient entered the asylum the skin round her neck was ecchymosed, the injury having been produced by a cord. Owing to her constant attempts at suicide it was necessary to put her in a straight-jacket. When she spoke, which was at rare intervals, she stated that she was accused of murder, and the police had come to arrest her. She rarely moved during the day, but at night was restless, and did not sleep. There was incontinence of urine and fæces. For fifteen days she had refused food. Complete recovery after a month's hypnotic treatment.

Dr. Répoud (Cantonal Asylum of Marsens, Fribourg) claims to have obtained results similar to those of Voisin in cases of insane patients who were not hysterical. Burckhardt (Préfargier Asylum) records successful cases of delusional insanity and acute puerperal mania; Liébeault, hereditary suicidal monomania; Dr. Brémaud (Brest), religious melancholia following confinement; Dr. Velander (Yonköping, Sweden), melancholia with mutism.

Dr. Robertson, while at Morningside Asylum, was able to hypnotise and control the worst case of suicidal and homicidal mania there had been in Morningside for ten years. He found hypnotism of much value in the management of violently insane patients.

In 1897, Dr. Woods, Hoxton House Asylum, reported good results in several cases of insanity and allied disorders.

Forel suggested that Voisin's so-called insane patients were not genuinely insane, and only suffered from aggravated forms of hysteria. Voisin denied this, but said that even if this were so, the successful treatment by suggestion of hysteria alone marked a therapeutic advance of no little importance. Our asylums, he said, contained a large number of hysterical insane who were violent and dangerous, and who inspired both disgust and pity by their tendency to drink and steal, and by their lies, dirtiness, and obscene and unnatural acts. Some of those who had long been asylum inmates were now, thanks to hypnotism, leading active and useful lives, a fact which he thought went far to prove the value of this form of treatment. Forel himself, at a later date, recorded a case of delusional insanity with hallucinations, which he had successfully treated.

Those unacquainted with hypnotic literature might be led by X's statements to conclude that hypnotism at home was represented by his failures and abroad by Forel's solitary dipsomaniac. The account I have just given, though the merest outline of hypnotic therapeutic work, is sufficient to indicate that X's picture of it is, to say the least, lacking in proportion and perspective.

PROCEEDINGS OF THE NEUROLOGICAL SOCIETY OF LONDON.

At the Clinical Meeting to be held on March 2, 1899, at 8.30 p.m., in the Medical Society's Rooms, 11, Chandos Street, the following cases were shown:—

Dr. F. E. Batten.—*Unusual form of muscular atrophy in a boy aged 5½ years.*

J. B., aged 5½; learnt to walk at two years of age, but never walked properly. He very easily becomes fatigued.

Present Condition.—Poorly-developed child with wasted limbs, especially below the knees and the forearms; contraction of muscles, giving rise to flexion of the legs at the hip and knee-joint and to flexion of the arm at the elbow; walks on tip-toe with toes inverted; unable to rise from the floor into the standing position, but can get on to his knees; all movements capable of being performed; no hypertrophy of muscles; knee-jerks present.

Dr. T. D. Savill.—*A case of epileptiform seizures of (supposed) vaso-motor origin.*

Mary M., aged 61, had been subject all her life, on and off, to three symptoms—"faintings," "flushings," and "nose-bleedings"—chiefly in her youth, and latterly, seven months ago, when she had had much trouble, the nose-bleedings ceased, and were suddenly replaced by the occurrence of more severe faints; and two or three months ago the fainting attacks *gradually* became attended by movements of the limbs until they assumed all the characters of idiopathic epilepsy, occurring several times daily, in some degree. Epigastric aura (which might be absent) followed by unconsciousness. The attack might or might not be attended by movements of the limbs. In one seen by Dr. Savill the patient, who was standing, gave an inarticulate cry, which was followed by a long inspiration. She seized hold of the screen which was near her, shook it violently, walked backwards with eyes fixed till she came to the wall, articulating as she did so "ub-ub-ub-ub-ub." The lips became somewhat purple, the face rather pale, and the pupils dilated. Saliva ran out of the mouth, and she made smacking movements with her lips as though tasting something. The respiration was laboured and the attack ended by a long expiration or sigh. The patient was quite unconscious, but the

whole seizure only lasted about sixty or seventy seconds. When the patient came round she expressed herself as feeling perfectly well, and there was no drowsiness. The heart was somewhat feeble, and probably both ventricles were somewhat dilated. A faint systolic apical murmur (? of dilatation) was heard when she first came to hospital, but had since disappeared. The other viscera and the urine are normal. It was suggested that this case appeared to support the theory of the vaso-motor origin of epilepsy, on account of the gradual transition of obviously vaso-motor phenomena into attacks which were to all intents and purposes idiopathic epilepsy, occurring for the first time in a woman aged 61.

Dr. H. A. CALEY.—*Case with symptoms of syringomyelia with sudden increase followed by rapid diminution in intensity.*

History.—W. H., aged 30, bricklayer. Rheumatic fever twice. No history of syphilis. Symptoms said to date from severe fall twelve months ago. Difficulty of walking gradually developed, followed by progressive weakness with numbness of right hand and arm. Marked disposition to sleep, occasional headache and vomiting, but neither severe. On Jan. 7, 1899, he had a "fit," in which he suddenly fell down unconscious. There were movements of head from side to side, but no clonic movements of extremities, arms crossed on the chest, with right hand tightly clenched. When the fit was over it was found that he could not stand or move his arms, which appeared quite helpless. Next morning was able to walk with difficulty. After this fit the previous symptoms were much intensified—marked weakness of both arms, "claw-like hand," with inability to move fingers on right side, legs unsteady and stiff.

Condition on admission.—Jan. 14, 1899. Marked weakness of right hand, with conspicuous wasting of thenar and hypothenar muscles and of interossei, 3rd and 4th being most affected. *R. D.* in right opponens and abductor pollicis, hypothenar muscles and 4th interosseus. First three interossei acted to faradism, but response increasingly feeble on passing to ulnar side. Also weakness with some atrophy (but no electrical changes), of right serratus magnus and thromboideus major. Scoliosis. Marked rigidity of legs with spastic gait. Sensation normal over lower extremities and trunk as high as 5th rib, above this tactile sensibility normal or slightly blunted, painful and thermal stimuli not recognised as such. This also applied to sides of neck, front of face, and upper extremities excepting a small area along inner side of left upper arm. Knee-jerks greatly exaggerated, foot clonus and rectus clonus. Sphincters unaffected. Treated with liq. hyd. perchlor. and pot. iodid.

Subsequent progress.—During the ensuing week decided improvement; could walk better, legs less rigid, more power in right hand and arm; sensation unchanged.

A week later thermo-anæsthesia and analgesia over the chest much less marked. One month after admission, *i.e.*, five weeks

after the fit, marked improvement in all respects. Weakness of right hand and arm less, gait comparatively normal, myotatic irritability less, thermo-anæsthesia and analgesia only partial in degree and much less extensive, being limited to sides of neck, first intercostal space, and arms.

Dr. WILFRED HARRIS.—*Hæmatomyelia.*

History.—A. J. B., aged 26, cook. Three and a half years ago, whilst bathing in the sea, experienced sudden pain across shoulders and down arms, and immediately after complete paralysis of both arms. Walked home quarter of a mile, and in another half-hour became completely paraplegic. Legs completely paralysed for a fortnight, then gradual improvement. Left arm began to improve after six weeks, and right arm in three months. Power over sphincters partially regained after two months.

P.S.—Slight spastic gait, drags left foot. Knee-jerks +. Complete wasting of right serratus magnus, latissimus dorsi, triceps, and lower half of pectoral and of upper portion of left pectoral, and slight wasting of left thigh and calf. Slight lateral curvature of spine.

Marked analgesia and thermanæsthesia over whole body and limbs up to middle of neck. Tactile sensation almost perfect. Now often burns himself unconsciously. Slight sphincter trouble.

Dr. J. H. BRYANT.—*Functional tremor closely simulating paralysis agitans, following typhoid fever.*

Male, aged 31. Engine-room artificer, R.N., suffering from tremor of fingers, hands, &c. He was quite well as far as his present complaint was concerned until convalescence from typhoid fever, for which he was admitted to a Hong-Kong hospital on July 2, 1898. About a month after being in the hospital he noticed shaking of his hands and legs. It was first detected in and always had been most marked in the fingers and thumb of the left hand. He had also noticed since his return that he got occasional hot flushes, and that he sweated a good deal at night. He had had electrical treatment for six weeks, and thought he was much better for it.

Present condition.—He has marked tremor of both hands, especially of the left. The thumb and index-finger are continually on the move. The tremor may be described as resembling the characteristic pill-rolling movement of paralysis agitans. When he raises himself from the supine position without using his arms he gets a rather violent tremor of the muscles of his body and limbs. There is no obvious loss of power in the hands or arms. His legs appear to be weaker than they should be. There is no loss of sensation. The knee-jerks are brisk. There is no ankle or patellar clonus. No atrophy of the muscles. No R. D. No oculo-motor paralysis. There is slight temporary oscillation of the eye-balls when they are moved in a lateral direction; he cannot keep his eyes fixed and looking to the left or right without a feeling of fatigue and strain.

Dr. STANSFIELD.—*Epileptic insanity associated with mastoid disease. Operation: Improvement of mental symptoms, and cessation of fits.*

S. H., aged 31, laundress, commenced to have a discharge from the right ear when twenty years of age. A year later she had her first fit. Epileptic fits continued at frequent intervals up to the date of the first operation, a period of nine years, during the last three months of which she had 154. For the latter half of the nine years she had grave mental symptoms. The discharge from the ear continued during this time, but was intermittent. On January 28, 1898, her mastoid cells were trephined. She had had no fit since the operation, but she had continual headache, sickness and vomiting, with puffiness and tenderness of the scalp to pressure over the right ear. Dean's operation was performed on April 12, 1898, and the parts thus exposed were explored. No signs of pus were found, but the operation was contemporaneous with the disappearance of the above symptoms. The grave mental symptoms passed off with the first operation, and the dementia, which was becoming pronounced, has gradually cleared. There had been no more fits.

Dr. H. H. TOOTH.—*A case of atrophy of the lower extremities of the peroneal family form.*

The patient, a male, aged 21, a painter, suffered at six years of age from weak ankles. This weakness progressed and resulted in the formation of pes cavus, which required tenotomy on several occasions. A year ago the hands became affected with atrophy of the intrinsic muscles, especially of the first and second dorsal interossei. The muscles of the thighs were wasted in their lower fourth, and those below the knees had almost disappeared, with loss of faradic irritability on electrical stimulation. There were five in the family besides the patient, who was the third child. Of these all except one were affected with some atrophy of the leg muscles with more or less marked pes cavus.

Please substitute this plate for Plate I. which appeared in the Spring Part, illustrating the paper on "A Form of Nerve Termination," by Dr. Aldren Turner and Dr. W. Hunter.

PLATE I.

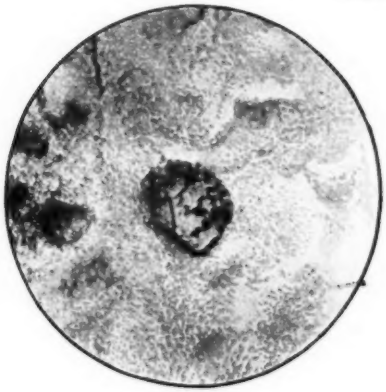

FIG. 1.

FIG. 2.

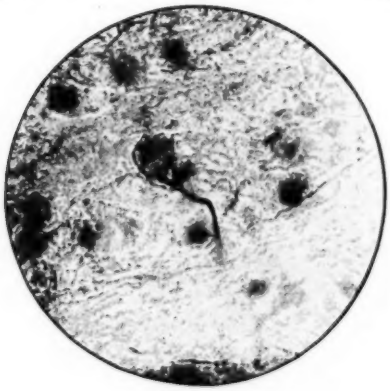

FIG. 3.

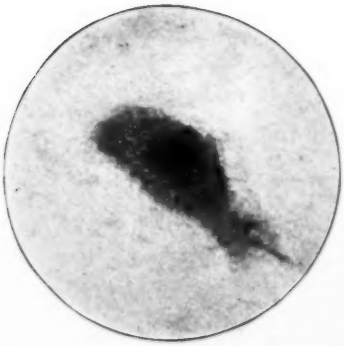

FIG. 4.

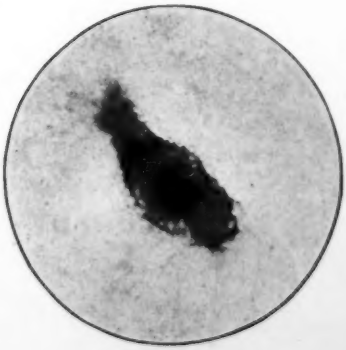

FIG. 5.